AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 22 JANUARY, 1939 NUMBER 1

FACIAL HEMIATROPHY*

A REPORT OF TWO CASES

FRANK B. WALSH, M.D.
Baltimore, Maryland

Facial hemiatrophy deserves mention in current ophthalmological literature because ocular changes, although usually inconspicuous, may be observed. The purpose of this paper is to report two cases and to indicate the present state of knowledge on the subject.

There are two important types of the disease: the congenital nonprogressive, and the more frequent progressive facial hemiatrophy. Congenital facial hemiatrophy has been recorded rarely. It is characterized by congenital hypoplasia with subsequent retardation of growth (Wartenberg[1]). Case 1 here reported presented a classical picture of this abnormality.

CASE 1

I. G. (104, 047), a white boy, age 13 years, was examined in the dispensary clinic of the Johns Hopkins Hospital in March, 1937. He complained of poor vision in the right eye noticed three months before his visit to the dispensary. The right side of his face has been flatter than the left since birth.

He was the fifth of seven children, all of whom were normal except the patient. His birth was normal. His mother noticed immediately that his right cheek was yellow and covered with fine hair and appeared flatter than the left. The upper right gum became swollen and sore at seven years of age, but no teeth had appeared. The unerupted deciduous teeth were later extracted.

Examination showed a facial asymmetry, the right side of the face being much flatter than the left. On the right side there was much lanuginous hair with atrophy of the muscles and wrinkling of the skin. The right outer canthus was displaced downwards. There was no motor paralysis nor sensory change in the involved region.

Examination of the right eye showed the lids, lacrimal apparatus, conjunctiva, cornea, anterior chamber, iris, tension, and external ocular movements to be normal. The pupil was round, regular, and reacted to light and on convergence, and was of the same size as that of the left eye. There was no enophthalmos. Ophthalmoscopic examination showed a large central pigmented chorioretinal lesion which involved the macula. The remainder of the fundus was normal. The left eye was normal throughout. Vision in the right eye was 10/200, in the left 20/15.

Roentgenograms showed that permanent teeth were present in the upper right gum. None had erupted. The frontal and maxillary sinuses and the basal foramina were smaller on the right than on the left. Figure 1 illustrates the appearance

*From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University. Read before the Section on Ophthalmology, Baltimore City Medical Society, February 24, 1938.

of the patient at the time of admission.

Progressive facial hemiatrophy is not an extreme rarity. J. Purves Stewart[2] described the disease as follows: "This disease, which commences in early life, usually before puberty, and more often in females than in males, shows itself first in the skin of the face, either near the orbit or over the upper or lower jaw, gradually spreading over the whole face on one side. The skin becomes thinned from atrophy of the papillary layer, the subcutaneous fat disappears, and thus the affected side of the face becomes wrinkled and furrowed in marked contrast with the healthy side. Later the subjacent muscles, cartilages, and bones become atrophied, but without motor paralysis or reaction of degeneration. The corresponding side of the tongue, when protruded, comes straight out, unlike that of a patient with atrophy from hypoglossal palsy. The hair on the affected side may fall out or become white, and the sebaceous glands may atrophy. The scalp rarely is affected. There is no anesthesia." This concise description suffices for many classical cases, but others are recorded in which more complicated pictures were present.

Progressive facial hemiatrophy has been observed as an accompaniment of scleroderma so frequently that it is considered by some authorities to be a definite form of that disease. Syringomyelia may result in either hemiatrophy or hemihypertrophy. Hemiatrophy has been reported as following epidemic encephalitis (Mankowski[3]). It has followed Klumpke's paralysis and infantile hemiplegia, but with these conditions this paper is not concerned.

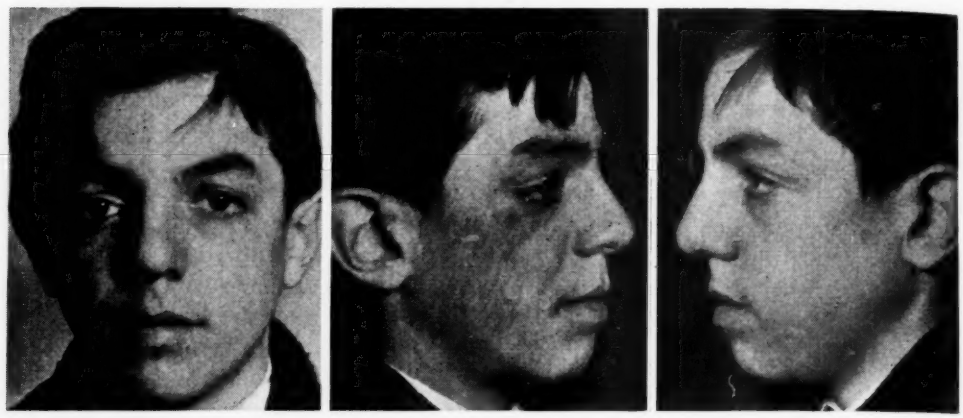


Fig. 1 (Walsh). Congenital (nonprogressive) facial hemiatrophy. Case 1.

Associated symptoms and signs have been: (1) contracture of muscles of the affected side of the face. Sachs's[4] patient exhibited clonic then tonic spasms of the temporal and masseter muscles which spread to the tongue on the atrophic side. Convulsive tic was noted by Oppenheim,[5] and intention tremor of the upper extremity on the same side by Claude.[6] (2) Hemicrania was observed by Oppenheim. (3) Jacksonian epilepsy has been observed in many cases (Wartenberg,[1] Wolff[7]). Calcification of the brain and epilepsy were recorded by Merritt, Faber, and Bruch.[8] These cases have some weight in pointing to a possible cerebral focus as the causative agent in the condition. (4) Recurrent herpes was

observed by Trotter.[9] (5) Tachycardia was noted by Jendrássik.[10] (6) Esophageal dilatation was recorded by Claude and Cantraine.[11] (7) Shumway[12] has reported the case of a patient who exhibited facial paralysis, optic neuritis, and facial hemiatrophy on a basis of polyneuritis. Facial hemiatrophy also has been observed with facial paralysis (Wartenberg[1]), but Gower[13] is authority that true facial hemiatrophy never results from facial paralysis. (8) Acromegaly was observed by Harbitz.[13a] (9) Pigmentation of the skin has been observed frequently. (10) Ocular changes have been noted frequently and are described separately.

Obviously progressive facial hemiatrophy is a misnomer when the process involves other parts of the body. It may commence in the upper triangle of the neck (O. Fischer[14]). The shoulder girdle on the same side of the body as the affected face, or the whole of one side of the body may be involved in the process (Oppenheim,[5] Finesilver and Rosow[15]). The face on one side may atrophy with a coincident atrophy of the trunk and limbs on the opposite side (Lunz,[16] Bernstein[17]), or a facial hemiatrophy may occur in association with pigmentation on the opposite side of the body (Volhard[18]). Bilateral hemiatrophy (Schlesinger) occurs not infrequently (Archambault and Fromm[20]).

Case 2 here reported is an example of progressive facial hemiatrophy, and is especially interesting in that the patient suffered from heart block, a complication hitherto unreported in facial hemiatrophy. Only the essential details of this case are presented.

CASE 2

L. C. (H.L.H. 72,374), a white girl, was first seen in June, 1931, at which time she was five years old. She was brought for admission because of a difference in the two sides of her face.

Past history: She was the third child of the family, having two brothers who were two and four years older, respectively. Both siblings developed normally. The parents were healthy without developmental defect. During her fifth year she contracted measles, chickenpox, and influenza. One month before examination in this hospital she had whooping cough. One year before her examination here she had been hospitalized elsewhere, at which time a diagnosis of heart block was made. She recovered from this after a few months. The cause was not ascertained.

Present illness: At the age of three years her mother noticed a bluish tint in the skin at the left corner of her mouth. Two or three months later it was observed that the left lip had become thinner than the right and was drawn outward. About this time a hollow above the left upper eyelid appeared. The whole left side of the face gradually became smaller than the right side. There was no complaint of twitchings of the facial muscles, of pain, or of abnormal sensations in the affected area. There was no visual defect.

Examination (June, 1931), age five years: The patient was a rather frail child with no complaints, with normal temperature and fast pulse (130). General physical examination revealed no abnormality other than that of the face.

The head was of normal shape and size. The face was asymmetrical. Above the left eye the forehead contour showed a large shallow depression about 3 cm. in diameter. The outline of this depression was definite and its walls sloped gradually inward. The face showed thinning of the upper lip on the side with thinning of the buccal mucosa. There was an indistinct line of pallor and atrophy 0.5 cm. wide

extending upward, just lateral to the left side of the nose and medial to the inner canthus of the left eye and merging with the atrophic area on the forehead. The veins of the left side of the face were not dilated. Over this area the skin was thin and freely movable over the underlying structures. The color of the skin was unchanged, but tiny dimpled areas were present in it over the involved areas. Palpation gave the impression that there was thinning of the underlying structures.

The eyes appeared to be normal, with pupils, equal in size, which reacted normally to light and on convergence. External ocular movements were normal. The ophthalmoscopic examination was negative, bilaterally.

Laboratory tests, including Wassermann reaction on the blood, were negative. Roentgenograms showed clouding of the left antrum.

Special tests: On this admission it was thought that the electrical resistance and the temperature of the skin were slightly higher on the affected side than on the normal side. Subsequently this difference was not found.

A diagnosis of facial hemiatrophy was made.

Second admission (1933), age seven years: The facial atrophy appeared more advanced. In addition, there was an abnormally slow pulse rate of 44. An additional change in the face was thinning of the soft palate on the left. The left maxilla had become much wasted. The nasal bones on the left side were smaller than those on the right. The upper left gum was thickened and the teeth in that region were irregular. The mandible and the tongue were not affected. There was no alteration of sensibility nor demonstrable weakness of the muscles. The cranial nerves remained normal.

General examination showed slight cardiac enlargement. The apex beat was in the sixth interspace 4.5 cm. outside the mammary line. The blood pressure was normal. Other examinations, includ-

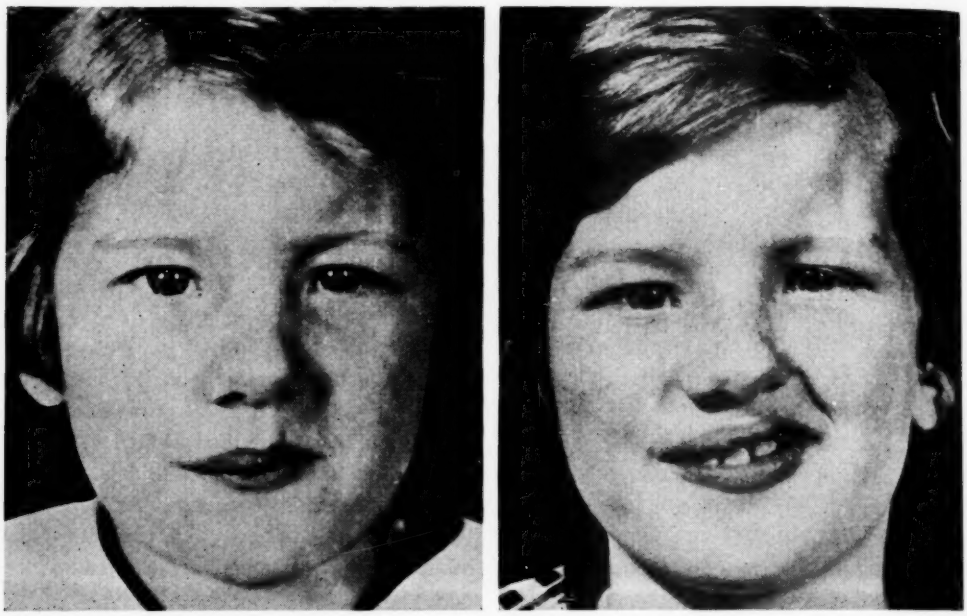


Fig. 2 (Walsh). Progressive facial hemiatrophy. Patient in case 2 at five and eight years of age.

ing laboratory tests, gave negative results.

Examination of the eyes showed them placed at different levels, the left eye appearing higher than the right, There was no diplopia. There was definite enophthalmos (not measured) of the left eye. No difference in the width of the lid slits was observed. The extraocular movements were normal. The conjunctivae and corneae were normal. The pupils were regular, of equal size, and reacted promptly to light and on convergence. Vision of the right eye 20/30; of the left eye, 20/30. The optic fundi were normal. Corrected vision O.U. was 20/20.

Further X-ray studies showed the left frontal sinus to be larger than the right. There was no apparent change in the bones of the skull. Blood sugar and sugar tolerance curves were normal. Phthalein excretion was 78 percent in two hours; N.P.N. was 39 mgm. percent; B.M.R. was —18.

Special tests: A series of tests was performed using pilocarpine (1/20 gr. in 10 minims of water). Pilocarpine injections produced perspiration under the armpits and flushing of the face on both sides. Local injections in the regions of the orbits caused profuse lacrimation of both eyes.

A series of electrocardiogram records was obtained. These showed a third-degree heart block (complete auricular-ventricular dissociation) which was not altered by repeated injections of atropine sulphate. The oculo-cardiac reflex showed a ventricular slowing from 45 to 41.

Interval 1933 to 1937: During this period there were no important new developments other than a gradual increase in the atrophic process. Heartblock was present constantly. The patient attended school. She was admitted and examined on two occasions, but nothing materially was added to the clinical picture.

Fifth admission (1937), age 11 years: She was readmitted in March with a history that four months prior to this admission she had had a spell of numbness of the left side of the face and right hand and inability to speak for a period of 15 minutes. This was followed by nausea without vomiting or headache. Following this attack she had frequent attacks of nausea with a sensation of numbness in the tongue and left hand. These later spells were followed by severe headache in the left temporal region. On one occasion while in the hospital she complained of a headache followed by a sensation of haziness of vision and numbness of the right hand and lower arm. Examination of the eyes showed no change from the examination made in 1933. She contracted diphtheria, and was discharged to another hospital.

Summary of history: A girl three years old developed leftsided facial atrophy which progressed during the six years she was under observation. When she was four years old heart block appeared, and was recovered from, but recurred three years later and persisted during the remainder of the period of observation. Eight years after the onset of the facial hemiatrophy spells of numbness in the face and superior extremities occurred. Extensive studies failed to establish cause for the heart block. Special tests designed to establish involvement of the sympathetic nervous system were negative.

Comment

Since Jarry (Chasanow)[21] in 1837 and Romberg[22] in 1846 described progressive facial hemiatrophy, the cause of the disease has remained obscure. A review of the theories of etiology summarizes the present knowledge on the subject.

The trigeminal peripheral-neuritis theory. Mendel[23] in 1889 examined a single case at autopsy, and found a peripheral

interstitial neuritis of the fifth nerve. He cautiously suggested that this might be the basic factor in initiating the disease process. Mendel's findings were later duplicated in another autopsy case (Loebel and Wiesel).[24] However, in 1914 Grabs[25] found no involvement of the trigeminal nerve or any part of the central nervous system.

Pain in the region of the fifth-nerve distribution is a frequent symptom and may be a precursor of hemiatrophy (Oppenheim).[5] However, typical trigeminal neuralgia is not followed by hemiatrophy. The trigeminal neuritis theory seems to be invalid because (1) section of the sensory root of the trigeminus does not result in facial hemiatrophy; (2) the atrophic process does not start invariably in, or remain in, the region of trigeminal innervation.

Although the trigeminal-neuritis theory as such appears invalid for the reasons stated above, it has been suggested that frequent limitation of the process to the region of the fifth-nerve innervation is due to involvement of the sympathetic fibers which accompany the fifth nerve. Thus it is thought to be a "trophoneurosis."

The sympathetic theory. Involvement of the cervical sympathetic probably through vasomotor control has been considered frequently as the cause of the condition (Sachs,[4] Cassirer,[26] Siebert[27]). Trauma has been recorded as an initiating factor (Bost[28]).

In the earlier work, involvement of the peripheral sympathetic system was considered principally. Oppenheim, in his excellent review, includes cases following tuberculous infections of the apex of the lung, extirpation of the glands of the neck, and direct injuries to the face and neck, all of which might affect the cervical sympathetic system. Manthey[29] described facial hemiatrophy following thyroidectomy and remarked that signs of sympathetic injury occurred only 12 times in 1,196 thyroid operations (Kaelin's series).

On the basis of the "trophic" nature of the lesions, many authors thought there was involvement essentially affecting the sympathetic system, either through the cervical sympathetics or central sympathetic tracts, including the parasympathetics. Stief[30] reported the autopsy findings in a case of right-sided, progressive hemiatrophy with scleroderma in a woman of 64 years (the oldest case observed) over a period of four years. There was generalized sclerosis of the central vessels, and the brain itself showed various stages of necrosis with an interesting difference in the two hemispheres. The left hemisphere in its entirety showed coagulative necrosis, apparently from anemia; whereas the right hemisphere showed vascular dilatation and stasis. This difference between the right and left halves was less marked in the brain stem than in the hemispheres. Stief also found round-cell infiltration in the right cervical ganglia. He thought that the vasomotor change in both hemispheres was dependent upon involvement of the cervical sympathetics—that on the right to vasomotor paralysis and that on the left to vasomotor irritation. He thought that the hemiatrophy in his case was due to destruction of the opposite thalamus.

As further case records accumulated, symptoms other than facial atrophy were noted, and it became apparent that involvement neither of the trigeminus nor of the sympathetic could adequately explain the entire picture.

Epileptic fits were noted by Wolff,[7] Wartenberg,[1] Archambault and Fromm,[20] Lauber,[31] and Merritt, Faber, and Bruch.[8] They usually commenced several years after the onset of the hemiatrophy. Case

2 here recorded is of interest in this regard.

The occurrence of fits, the occasional extensive spread of the process (Wolff,[7] Archambault and Fromm[20]), coincident cerebral calcification (Merritt, Faber, and Bruch[8]), ocular signs such as pupillary dilatation with loss of reflex to light (Oppenheim,[5] Claude and Cantraine[11]), monocular nystagmus (Langelaam[32]), and ocular muscle palsies (Heiligenthal,[20] Sterling[31a]) suggested central lesions. Crossed lesions in which the face on one side and the limbs and trunk on the opposite side were affected suggested midbrain lesions.

Involvement of the pyramidal tracts as reported by Claude appeared to indicate central involvement.

The infection theory. Many cases have been described as occurring after infectious diseases, and Möbius (Oppenheim[5]) thought that progressive hemiatrophy was due to some infectious process. Infection from the tonsils or following adenoidectomy has been mentioned.

Heredity of facial hemiatrophy. Klingmann[33] in this country described a family in which the grandmother, mother, and her twin daughters showed the defect. degeneration, and remarked that, in common with other such diseases, it often makes its appearance at the time of puberty. He further stated that, as in the other degenerative diseases, it may be accompanied by imbecility, congenital paralysis of the eye muscles, facial paralysis, and other conditions. However, heredity is not stressed by most authors.

Relationship between progressive facial hemiatrophy and scleroderma. It generally is agreed that differentiation between these two diseases may be difficult, if not impossible, particularly at the onset. Scleroderma does not usually occur unilaterally, but it may do so, particularly in the circumscribed form (morphea). Some authorities (Cassirer,[26] Knapp[34]) consider scleroderma and hemiatrophy to be manifestations of the same disease process. The small circumscribed patch in the skin which denotes the commencement of scleroderma usually is raised and often is of a slightly bluish or brown color, but may be depressed and free of pigment. Oppenheim, in speaking of facial hemiatrophy, stated that there might be an infiltration of the skin before the atrophy appeared, and Wartenberg described a case in which there was scarcely any involvement of the skin. Pigmentation has been observed in both diseases. For a careful differentiation the reader is referred to Osborne's[35] article.

The groove lying lateral to the midline in characteristic progressive facial hemiatrophy may contain hard immobile skin as in scleroderma. Pick[36] described such a case, diagnosed originally as scleroderma because of the characteristic appearance of the skin, which developed into a progressive facial hemiatrophy.

In other cases, however, the hard, shiny, thickened, immovable skin of the scleroderma patient scarcely can be confused with the smooth, thin, freely movable skin of the progressive-facial-hemiatrophy patient. The cause of scleroderma is as obscure as that of progressive facial hemiatrophy, and the same theories in regard to etiology have been put forward for each disease.

Ocular signs of hemiatrophy. Enophthalmos, sometimes of a high degree, is occasionally a striking result of progressive hemiatrophy whether or not it is associated with scleroderma. It was present in case 2 here reported. However, it has not been noted in the majority of cases. Exophthalmos has been observed.

Apparent upward displacement of the eye on the affected side was observed by Archambault and Fromm and was pres-

ent in case 2 here reported, but in neither instance was there an abnormal muscle balance.

Narrowing of the lid slit on the affected side has been observed frequently, but was not present in the case here reported. Chasanow[21] reported a case which presented Stellwag's sign (infrequent blinking).

The extraocular muscles rarely are affected. Salomon[37] described the case of a girl of nine years who possibly had congenital syphilis, and in whom there was a weakness of the facial muscles and atrophy on the left side of the face. There was also paralysis of the right abducens, and partial paralysis of the third nerves innervating the internal recti. The vision was relatively good. There were no pupillary reactions to light nor to convergence on either side. Lacrimation was normal. Sterling[31a] also reported abducens paralysis. Finesilver and Rosow[15] recently reported a case of complete unilateral hemiatrophy with enophthalmos, slight lagophthalmos, atrophy of the nasal half of the upper margin of the lid, contraction of the conjunctival sac, and impairment of all extraocular movements. Monocular nystagmus was reported by Langelaam.[32] Heterochromia iridis has been reported (Lauber,[31] Pick[36]).

Pupillary signs have been recorded, but are not uniform. The pupil may be dilated and fixed to light on the same side as the atrophy (Stief,[30] Archambault and Fromm,[20] Oppenheim[5]) or on the opposite side (Langelaam[32]). In several instances where the pupil was narrowed, paralysis of the sympathetic fibers was evidenced by failure of the pupil to dilate with cocaine or adrenalin (Stief,[30] Stiefler[38]). But in many instances the pupillary reactions were normal. Accommodation may be affected (Archambault and Fromm[20]).

Vision was not affected in the great majority of the reported cases. Optic atrophy was noted in a few cases (Heiligenthal and Abadie quoted by Surat,[39] Claude and Cantraine[11]).

Summary of ocular signs

Ocular signs are not constantly observed in facial hemiatrophy. When present, the principal ones are enophthalmos, rarely exophthalmos, narrowing of the lid slit, and pupillary changes. It is noteworthy that Horner's syndrome in its entirety is rather a rarity, and miosis, which is the most definite indication of an injury to the sympathetic chain, seems to occur no more frequently than does dilatation of the pupil. The latter may be ascribed in these cases to injury to the parasympathetics. However, the dilated pupils may be examples of "tonic" pupils and, therefore, have no known significance. Salomon's case loses some of its value because of the possible presence of congenital syphilis, but would indicate an extensive central lesion. Monocular nystagmus has been reported only once, and was not clear cut in that case. Heterochromia iridis has been observed rarely. It would seem to indicate sympathetic involvement during an early period of life.

The function of the eye usually is not affected.

Cardiac action in progressive facial hemiatrophy. Several authors, among them Jendrássik,[10] noted a persistent increase in the pulse rate. Case 2 here reported is, so far as I am aware, the only case report of facial hemiatrophy associated with heart block. This association is probably coincidental.

The special tests in progressive facial hemiatrophy. Tests designed to reveal hyper- or hypoactivity of the sympathetic nerves have been performed in many cases, but the results were not uniform.

The most important tests are: (1) co-

caine instillations, which fail to dilate the pupil when sympathetic innervation is interrupted, or (Stiefler[38]) accelerate dilatation when the sympathetics are stimulated; (2) pilocarpine injections, which fail to produce sweating on the area in which there is loss of sympathetic innervation; (3) temperature estimations when there is elevation of temperature in the area of sympathetic loss. In case 2 here recorded the two last-mentioned tests were negative.

Interesting conditions described in the literature may be associated with or related to facial hemiatrophy. These are: (1) hemihypertrophy, which occurs less frequently than hemiatrophy but has been reported as occurring in association with it (Sterling[20]). (2) Lipodystrophy as described by Ziegler.[40] (3) A symptom complex described by Passow[41] under the title "Horner's syndrome, heterochromia, and status dysraphicus." Passow's paper has recently been reviewed adequately by Pino, Cooper, and Van Wien.[42] (4) A syndrome is described by Fuller, Albright, *et al*[43] under the title "Syndrome characterized by osteitis fibrosa disseminata, areas of pigmentation, and endocrine dysfunction with precocious puberty in females."

Conclusion

Two cases of facial hemiatrophy have been described. One was congenital, and for this reason merits recording because of its rarity. The other case deserves recording because the combination of progressive facial hemiatrophy and heart block has not been reported previously.

Bibliography

[1] Wartenberg, R. Zur Klinik und Pathogenese der Hemiatrophia faciei progressiva. Arch. f. Psychiat., 1925, v. 74, p. 602.

[2] Purves-Stewart, J. The diagnosis of nervous diseases. Ed. 5, New York, E. B. Trent & Co., 1920, p. 142.

[3] Mankowski, B. N. Quoted by Archambault and Fromm.[20]

[4] Sachs, B. Progressive facial hemiatrophy with some unusual symptoms. Med. Rec., N.Y., 1890, v. 37, p. 292.

[5] Oppenheim, H. Lehrbuch der Nervenkrankheiten, 6. Aufl., Berlin, S. Karger, 1913, p. 1792.

[6] Claude, M. Blessure carotidienne droit, par éclat de V.B. Syndrome medullo-sympathique caractérise hémiatrophie faciale gauche et des troubles vasculaires du membre supérieur du meme côte. Mydriasis gauche paradoxicale. Tremblement du bras gauche a type de sclérose en plaques. Rev. Neurol. 1927, v. 1, p. 244.

[7] Wolff, H. G. Progressive facial hemiatrophy; report of case, with other signs of disease of central nervous system. Arch. Otolaryng., 1928, v. 7, p. 580.

—— J. Nerv. & Mental Dis. 1928, v. 69, p. 140.

[8] Merritt, K. K., Faber, H. K., and Bruch, H. Progressive facial hemiatrophy. Jour. of Pediat., 1937, v. 10, p. 374.

[9] Trotter, W. Recurrent herpes zoster of the face with hemiatrophy. Proc. Royal Soc. London (Sect. Dermat.) 1915, v. 9, p. 43.

[10] Jendrássik, E. Ueber die Hémiatrophie faciei. Deutsches Arch. f. klin. Med., 1897, v. 59, p. 222.

[11] Claude, H., and Cantraine, E. Hémiatrophie faciale par lesion du sympathique cervical et dilatation primitive de l'oesophage. Rev. Neurol., 1918, v. 25, p. 187.

[12] Shumway, E. A. Association of optic neuritis, facial paralysis, and facial hemiatrophy. Arch. of Ophth., 1935, v. 13, p. 8.

[13] Gowers, Sir. W. R. The influence of facial hemiatrophy on the facial and other nerves. Rev. Neurol. and Psychiat., 1906, v. 4, p. 1.

[13a] Harbitz. Quoted by Archambault and Fromm.[20]

[14] Fischer, O. Quoted by Oppenheim.[5]

[15] Finesilver, B., and Rosow, H. M. Total hemiatrophy. Jour. Amer. Med. Assoc., 1938, v. 110, p. 366.

[16] Lunz. Quoted by Oppenheim.[5]

[17] Bernstein, E. Hemiatrophia alternans facialis progressiva mit halbseitiger Alopezia, Pigmentverschiebung und Hautatrophie. Dermat. Woch., 1930, v. 90, p. 235.
[18] Volhard. Quoted by Oppenheim.[5]
[19] Schlesinger, A. Ein Fall von doppelseitiger, umschriebener Gesichtsatrophie. Arch. f. Kinderh., 1905, v. 42, p. 374.
[20] Archambault, L., and Fromm, N. K. Progressive facial hemiatrophy; report of 3 cases. Arch. Neurol. and Psychiat., 1932, v. 27, p. 529.
[21] Chasanow, M. Beiträge zur Aetiologie der Hemiatrophie des Gesichts. Zeit. f. d. ges. Neurol. u. Psychiat., 1932, v. 140, p. 473.
[22] Romberg, M. H. Klinische Ergebnisse. Berlin, 1846, p. 75.
[23] Mendel, E. Quoted by Archambault and Fromm.[20]
[24] Loebel and Wiesel. Quoted by Oppenheim.[5]
[25] Grabs, E. Ein Fall von Hemiatrophia faciei progressiva. Neurol. Centralbl., 1914, v. 33, p. 85.
[26] Cassirer, R. Die vasomotorisch-trophischen Neurosen. Centralbl. f. Nervenh. u. Psychiat., 1900, n.F., v. 11, p. 591.
[27] Siebert, H. Ein Fall von rechtseitiger Gesichtsmissbildung mit Erscheinungen der Hemiatrophia faciei. Deutsche Zeit. f. Nervenh., 1917, v. 56, p. 320.
[28] Bost, C. Progressive facial hemiatrophy; report of case in child. Arch. Pediat., 1927, v. 44, p. 497.
[29] Manthey, P. Hemiatrophia faciei dextra nach Strumektomie. Zeit. f. d. ges. Neurol. u. Psychiat., 1928, v. 114, p. 192.
[30] Stief, S. Ueber einen Fall von Hemiatrophie des Gesichtes mit Sektionsbefund. Zeit. f. d. ges. Neurol. u. Psychiat., 1933, v. 147, p. 573.
[31] Lauber, H. Ein Fall von Hemiatrophia facialis progressiva mit Beteiligung des Auges. Zeit. f. Augenh., 1925, v. 57, p. 492.
[31a] Sterling. Quoted by Archambault and Fromm.[20]
[32] Langelaam, J. W. Sur un cas d'hémiatrophie faciale avec signe d'Argyll Robertson contralatéral. Rev. Neurol., 1913, v. 21, pt. 2, p. 520.
[33] Klingmann, T. Facial hemiatrophy. Jour. Amer. Med. Assoc., 1907, v. 49, p. 1888.
[34] Knapp, P. C. A case of hemiatrophy from scleroderma. Boston Med. and Surg. Jour., 1911, v. 164, p. 671.
[35] Osborne, E. D. Morphea associated with hemiatrophy of the face. Arch. Dermat. and Syph., 1922, n.s., v. 6, p. 27.
[36] Pick, W. Sclérodermie en coup de sabre mit osteoporotischer Zone im Stirnbein oder Hemiatrophia faciei? Arch. f. Dermat. u. Syph., 1933, v. 167, p. 543.
[37] Salomon, S. Ein Fall von Hemiatrophia progressiva mit Augennervensymptomen. Neurol. Centralbl., 1907, v. 25, p. 846.
[38] Stiefler, G. Ueber die Hemiatrophia faciei progressiva bilateralis. Jahrb. f. Psychiat. u. Neurol., 1934, v. 51, p. 277.
[39] Surat, W. S. Ueber einseitige Störung der Gesichtstrophik (Hemiatrophia faciei). Monatschr. f. Psychiat. u. Neurol., 1930, v. 77, p. 202.
[40] Ziegler, L. Lipodystrophies; report of 7 cases. Brain, 1928, v. 51, p. 147.
[41] Passow, A. Hornersyndrom, Heterochromie und Status dysraphicus, ein Symptomenkomplex. Arch. f. Augenh., 1933, v. 107, p. 1.
[42] Pino, R. H., Cooper, E. L., and Van Wien, S. Arachnodactyly and status dysraphicus; review. Ann. Int. Med., 1937, v. 10, p. 1130.
[43] Albright, F., Butler, A. M., Hampton, A. O., and Smith, P. Syndrome characterized by osteitis fibrosa disseminata, areas of pigmentation and endocrine dysfunction, with precocious puberty in females. New England Jour. Med., 1937, v. 216, p. 727.

STAPHYLOCOCCUS TOXIN*

An experimental study in rabbits

J. H. Allen, M.D. and A. E. Braley, M.D.
Iowa City

Toxins prepared from staphylococci isolated from general sources have been shown to exhibit several activities,[1-5] lethal, hemolytic, leucocytolytic, dermonecrotic, enterotoxic and perhaps others. Several of these properties have been shown for filtrates prepared from staphylococci isolated from ocular sources.[6, 7] This study was instituted in a further effort to correlate the properties of ocular types of staphylotoxin with those of general types. However, early in the problem normal rabbits were found to vary in susceptibility to the toxin. This phenomenon[8] has been observed by other workers. Ramon, Richou, and Descazeaux[9] have shown that a considerable proportion of normal rabbits possess "natural" antitoxin in their serum. This has been confirmed by Roy.[10] However, Glynn[11] has suggested that variation in potency of the toxin also may account for the so-called variation in susceptibility of normal rabbits. Therefore, in this study the antitoxin titer of the serum of the rabbits was determined, and the potency of the toxins was titrated against standard toxin and antitoxin furnished by the National Institute of Health.

Methods and materials

The 10 strains of staphylococci employed in making the toxins were isolated from cases of conjunctival inflammation. These organisms produced a yellow pigment on plain and blood agar, a yellow or purple growth upon crystal violet agar,[12] and produced acid in mannite media. Each strain caused coagulation of citrated human serum[13] and produced hemolysis upon both sheep's-blood agar and rabbit's-blood agar.

Toxin was prepared from each strain of organisms by a modification of the method of Leonard and Holm[14] as previously described by the senior author.[6] Just prior to administration of the toxin, it was titrated by the hemolytic method against standard toxin and antitoxin.

The provisional standard unit of toxin, an Lh dose, has been defined as that amount of toxin which, when mixed with one standard unit of antitoxin, will cause approximately 20-percent hemolysis of 1.0 c.c. of a 2-percent solution of packed red blood cells (rabbit). The toxins prepared for these experiments were standardized in Lh units and found to range between 8 and 10 per cubic centimeter. The Lh unit, however, was too large for estimations of the "natural" antihemolytic titer of rabbit serum; therefore, a subdivision was sought. A minimal hemolytic dose (MHD) was adopted—one which would just produce complete hemolysis of 1 c.c. of a 1-percent solution of packed red blood cells. Under the test conditions one Lh unit of the standard toxin was found to contain 200 MHD.

One hundred normal rabbits weighing from 0.8 to 3 kilograms were inoculated with amounts of toxin varying from 160 to 1,500 MHD (200 to 500 MHD per kilogram). The serum of each of 70 rabbits was titrated for its antihemolytic activity. Varying dilutions of serum were

*From the Department of Ophthalmology, State University of Iowa, College of Medicine. Part of a study being conducted under a grant from the John and Mary R. Markle Foundation. Read before the Association for Research in Ophthalmology in San Francisco, June 14, 1938.

added to tubes containing 2 MHD (1/100 Lh dose) of toxin; the volume in each tube was made up to 2 c.c. with normal saline, and the mixtures were incubated in a constant-temperature water bath at 37°C. for one hour. One cubic centimeter of a 1-percent solution of packed rabbit red blood cells, which had been washed in saline three times, was added to each tube. The tubes were left in the water bath for one hour, then placed in a cold room over night. Control tubes containing standard toxin and antitoxin were set up with each titration. In this manner it was possible to estimate antihemolytic activity to 1/200 unit. However, titrations to this fraction are not accurate because of the principle of neutralization in multiple proportions, and some workers[15] believe that below the level of 1/10 unit per cubic centimeter of serum the reaction is not specific.

In the beginning of the study the rabbits were given 200 to 500 MHD per kilogram in the marginal ear vein; as some of these survived, serologic studies were instituted. For this purpose approximately 10 c.c. of blood were drawn from one of the large superficial neck veins and 24 to 48 hours later toxin was administered intravenously.

A complete post-mortem examination was made immediately after death of the rabbit. Tissues were fixed in Zenker's fluid, sectioned in paraffin and stained by hematoxylin and eosin.

Serologic findings

The serum of 16 of the 70 rabbits was found to contain antihemolysin; however, only 9 contained an amount sufficient to protect against the toxin administered. Five rabbits possessing 0.5 units of antitoxin per cubic centimeter were protected against doses of 500 MHD per kilogram. The rabbits possessing less than 0.1 unit per cubic centimeter apparently were not protected against the toxin inoculations, thus perhaps lending support to the view that with such small amounts the reaction is not specific.

Pathologic findings

The survival time varied inversely with the size of the dose of toxin per kilogram of body weight. The rabbits receiving the larger doses died in 3 to 5 minutes and those receiving the smaller doses died in 30 to 60 hours.

Rabbits dying in 3 to 5 minutes usually had one short violent convulsion ending in a high-pitched squeal. Those surviving for a longer period exhibited symptoms of restlessness, increased rate of respiration, weakness, and passage of urine and feces. After lying quietly on the floor for a time, convulsive running movements developed and after one or more high-pitched squeals, the animals died.

TABLE 1

Antitoxin titrations

No. of Rabbits	Anti-hemolysin per c.c. of Serum	Dose of Toxin per Kg.	Fate
41	Not titrated	200–500 MHD	Died
5	Not titrated	200–500 MHD	Survived
5	0.5 unit	500 MHD	Survived
2	0.2 unit	500 MHD	Survived
2	0.1 unit	200 MHD	Survived
2	0.02 unit	200 MHD	Died
5	0.01 unit	200 MHD	Died
54	0.00	200–500 MHD	Died

In order to minimize the possibility of post-mortem tissue changes, autopsies were done immediately after the squeal. Heart action had ceased except for some fibrillation of the auricles; however, peristaltic movements usually continued for several minutes.

Gross pathology

Rabbits dying within 30 minutes after administration of the toxin exhibited very little gross pathology, but as the survival time increased the amount of congestion, hemorrhage, and necrosis increased. There was a variable amount of serous fluid in the pericardial sac. Usually the

left ventricle was in systole and the right in diastole. There were occasional subpericardial petechial hemorrhages in the animals surviving for longer periods. Petechial hemorrhages and small hemorrhagic areas were present in the lungs, the number and extent being in direct proportion to the time of survival. In the smaller rabbits there were superficial hemorrhages in the thymus. The liver was purplish brown, distended by an active congestion, and an occasional small subcapsular hemorrhage was seen. The spleen also was distended and purplish brown. The kidneys were most constantly affected, but here again the acute deaths were characterized by the least gross pathology. The kidneys of rabbits dying in less than 30 minutes were acutely congested and in animals surviving longer hemorrhages were present beneath the capsule. As the survival period increased the kidneys became mottled in appearance, due to yellowish-white areas of necrosis surrounded by purplish areas of hemorrhage. The capsule could readily be stripped from these areas. The adrenals were slightly congested in the acute deaths, but were mottled by hemorrhage and necrosis in the prolonged deaths. The bladder invariably was distended. There was no evidence of gross pathology in the gastrointestinal tract in any of the animals.

Ophthalmoscopic examinations were made shortly after death in approximately half of the rabbits, but no gross pathology was observed.

Microscopic pathology

Heart: There were a few petechial hemorrhages in the epicardium, myocardium and endocardium of rabbits surviving for longer periods. In addition there was an occasional small zone of necrosis of the muscle fibers.

Lungs: In the acute deaths there were dilatation and congestion of the capillaries with an occasional interstitial and subpleural petechial hemorrhage. With an increase in the survival time, the microscopic pathology increased; there were red blood cells in the alveoli in increasing numbers, and in the more prolonged deaths there were some patchy areas of necrosis of the alveolar epithelium.

Thymus: An occasional small subcapsular hemorrhage was observed in the thymus of the smaller rabbits surviving for several hours.

Liver: In the animals dying in less than 30 minutes, the liver showed dilatation of the sinusoids and central vein of the lobule, with some cloudy swelling of the liver cells. However, as the survival time increased, patchy areas of necrosis developed about the central vein of the lobule. In rabbits surviving from 30 to 60 hours the necrosis had extended outward to include all of the cells of the lobule and practically all of the lobules were affected. In a few of the rabbits there was some evidence of regeneration around the portal spaces, with the cells appearing to arise from the bile ducts.

Spleen: The spleen, like the liver, exhibited only congestion and dilatation in the more acute deaths. The sinuses were filled with red and white blood cells as well as with large numbers of mononuclear cells. There was an increase in the number of brownish pigment granules in the phagocytic cells and in the amount of free pigment in the sinuses, indicating increased hemolysis. In the animals surviving for longer periods there was some necrosis of the cells lining the sinuses and some disintegration of the pulp.

Kidney: The microscopic changes in the kidneys were the most constant. The changes after rapid death consisted chiefly of congestion and dilation of the arterioles and glomerular capillaries. However, these were of a patchy nature, for some portions of a section showed normal arterioles and glomeruli. As the survival

time increased, interstitial and subcapsular hemorrhages appeared, and there apparently were some hemorrhages into Bowman's capsule.

In those animals surviving the inoculation for 4 to 6 hours some swelling and degeneration of the epithelium of the convoluted tubules was observed. Those surviving longer showed more extensive hemorrhages, precipitates in the tubules, and in some regions dilatation of Bowman's spaces with albuminous precipitates. At this stage some of the nuclei of the tubular epithelium were pale staining, others were pyknotic. Later (24- to 60-hour survival) the nuclei had disappeared in large portions of a section, this area being surrounded by a bluish-staining zone apparently made up largely of nuclear débris, and immediately around this was a zone of hemorrhage.

Adrenal: There was a similar time relationship in the pathology of the adrenals. First there was congestion, then followed hemorrhages and necrosis. The necrosis usually involved the medulla then the cortex. The rabbits surviving 30 to 60 hours showed almost complete necrosis of the adrenals.

No microscopic changes were observed in the gastrointestinal tract or bladder.

Microscopic examination of the eyes revealed one fairly extensive choroidal hemorrhage, but no other pathology.

Discussion

The pathology produced in rabbits by these ocular strains of staphylotoxin was essentially the same as has been described[16] for staphylotoxin from other sources. The only difference was the failure of these strains to produce changes in the gastrointestinal tract as described by Rigdon.[17]

The amount of pathology in the individual animal varied inversely with the size of the dose of toxin and directly with the survival time. Rabbits dying rapidly showed very little pathology, those surviving for a few hours exhibited patchy zones of pathology, and those surviving for several hours presented more generalized changes. This phenomenon was expected, the typical action of a potent toxic material.

The potency of the toxin was estimated in hemolytic units shortly before its use, thus controlling any variation in the toxin. Therefore as the individual dose was measured in hemolytic units, and as the pathology and the survival time were proportional to the size of the dose, it may be concluded that the hemolytic unit was a satisfactory standard for the ocular strains of staphylotoxin.

As a further control upon the pathogenic action of the toxin, the antihemolysin content of the serum of 70 of the rabbits was estimated. Nine rabbits possessing 0.1 to 0.5 units of antitoxin per cubic centimeter of serum were protected against the usual doses of toxin. This confirmed the observations of Ramon[9] that a considerable proportion of normal rabbits possess "natural" antitoxin. It also explained the failure of comparable doses of potent toxin to kill all the rabbits in the preliminary experiments. Therefore it was concluded that all staphylotoxin studies on rabbits should be controlled by serologic studies.

Conclusions

1. The pathogenic action of ocular strains of staphylotoxin is similar to that of general strains, with perhaps the exception of the action upon the gastrointestinal tract.

2. The hemolytic unit apparently is satisfactory for estimating the potency of ocular staphylotoxin.

3. Serologic studies should be made upon all rabbits used for experimental study with staphylococci and their toxins.

References

[1] Burnett, F. M. The exotoxins of Staphylococcus pyogenes aureus. Jour. Path. and Bact., 1929, v. 32, pp. 717-734.

[2] Gengou, O. Contribution a l'étude des antigenes et des anticorps staphylocciques. Ann. d. l'Inst. Pasteur, 1932, v. 48, pp. 135-143.

[3] Weld, J. T. P., and Gunther, A. Differentiation between certain toxic properties of filtrates of hemolytic Staphylococcus aureus. Jour. Exper. Med., 1931, v. 54, pp. 315-322.

[4] Bigger, J. W. The production of staphylococcal haemolysin with observations on its mode of action. Jour. Path. and Bact., 1933, v. 36, pp. 87-114.

[5] Glenny, A. T., and Stevens, M. F. Staphylococcus toxins and antitoxins. Jour. Path. and Bact., 1935, v. 40, pp. 201-210.

[6] Allen, J. H. Staphylococcic conjunctivitis: Experimental reproduction with staphylococcus toxin. Amer. Jour. Ophth., 1937, v. 20, pp. 1025-1030.

[7] Thygeson, P. Bacterial factors in chronic catarrhal conjunctivitis. Arch. of Ophth., 1937, v. 18, pp. 373-387.

[8] Rigdon, R. H., Joyner, A. L., and Ricketts, E. T. A study of the action of a filterable staphylococcal toxin on the kidneys of normal rabbits. Amer. Jour. Path., 1934, v. 10, pp. 425-434.

[9] Ramon, G., Richou, R., and Descazeaux, J. L'antitoxine staphylococcique d'origine naturelle, chez l'homme et chez différentes espèces des animales. Rev. d'Immunol., 1935, v. 1, pp. 401-414.

[10] Roy, T. E. Cited by Glynn, see reference 11.

[11] Glynn, J. H. The pathogenesis of cortical necrosis of the kidneys in rabbits following the injection of staphylococcus toxin. Amer. Jour. Path., 1937, v. 13, pp. 593-603.

[12] Chapman, G. H., and Berens, C. Crystal violet agar as a differential medium for staphylococci. Jour. Bact., 1935, v. 32, pp. 199-205.

[13] ——. (a) Coagulase and hemolysin tests as measures of the pathogenicity of staphylococci. Jour. Bact., 1934, v. 28, pp. 343-363.

——. (b) The differentiation of pathogenic staphylococci from nonpathogenic types. Jour. Bact., 1938, v. 35, pp. 311-333.

[14] Leonard, G. F., and Holm, A. A method for the production of staphylococcus toxin and toxoid. Jour. Immunol., 1935, v. 29, pp. 209-221.

[15] Levine, B. S., and Dyer, R. E. Personal communication.

[16] Dolman, C. E. Pathogenic and antigenic properties of staphylococcus toxin. Canadian Public Health Jour., 1932, v. 23, pp. 125-132.

[17] (a) Rigdon, R. H., and Leff, W. A. A gastro-intestinal lesion associated with staphylococcic infection in man. Arch. of Path., 1936, v. 21, pp. 298-307.

(b) Rigdon, R. H. Production of colitis in the dog with staphylococcus toxin. Arch. of Path., 1937, v. 23, pp. 634-640.

Discussion

Dr. Edward Jackson (Denver): In listening to the paper, this question occurred to me: Was any examination made of the central nervous system, or was there any manifest reason for the rapid death of the rabbits that succumbed before the viscera had given evidence of disease processes from the toxin?

Dr. Allen: The examinations of the central nervous system were not mentioned, because microscopic examinations have not been completed. Gross pathology in the central nervous system was not different from that of the other organs. The rabbits dying acutely exhibited very little pathology. Those which lived from 6 to 30 hours showed acute necrosis and a few petechial hemorrhages over the surface of the brain. In one rabbit a rather extensive subdural hemorrhage was noted. The few microscopic examinations that have been completed add nothing further. However, we believe the congestion, hemorrhages, and acute necrosis are similar to those found in the remainder of the organs and are sufficient to account for death.

THE INFLUENCE OF THE CENTRAL NERVOUS SYSTEM ON THE PIGMENT MIGRATION IN THE RETINA OF THE FROG*

HERMANN M. BURIAN, M.D.
Hanover, New Hampshire

In the series of objective changes which occur in the frog's retina under the influence of light, one of the most obvious is the migration of the pigment of the retinal epithelium. If a frog has been kept in complete darkness long enough, so that full dark adaptation has been achieved, the black-brown grains of the pigment are found in the bodies of the cells, outside of the layer of the rods and cones. In the retinas of light-adapted frogs, however, the bulk of the pigment is scattered within preëxistent cellular processes (Angelucci[1]), between the rods and cones, reaching inward as far as the external limiting membrane. This migratory phenomenon is most evident in amphibia and fishes; it is less distinct in birds, and still less in reptiles. In mammals it can hardly be demonstrated.

Since its discovery by Boll[2] and Kühne[3] in 1877, the reaction has been the subject of numerous studies. Outstanding among the older ones are the investigations by Engelmann[4] and his pupil Van Genderen Stort[5] who discovered another important functional change in the frog's retina, due also to the influence of light, the contraction of the cones.

Engelmann has been especially concerned with the influence of the central nervous system upon the migration of the pigment and the contraction of the cones and has reached the conclusion that such an influence must exist. The evidence he had was manifold. He found that in frogs and doves the reaction took place even when only one eye was exposed to light, while the other was tightly covered and shielded from any illumination. This, however, happened only if the brain of the experimental animal was intact. After decerebration there was no reaction in the fellow eye. From this Engelmann inferred that there is an association between the cones and the pigment cells of the two eyes. Such a connection could be established only by the optic nerve, which, therefore, according to Engelmann, must contain centrifugal, retinomotor fibers. Another reflex migration of the retinal pigment can be observed in the frog when only the skin of the body is exposed to light, while a light-tight cap covers the head of the animal. Here again the pigment always assumes light position; the contraction of the cones is less evident. Finally, Engelmann could state that dark-adapted frogs, in which a strychnine tetanus was produced, showed totally expanded retinal pigment. Curare did not influence the position of the pigment. Stimulation of the eyes of dark-adapted frogs *in vivo* and *in vitro* with alternating induction currents of medium density produced the same effect.

Angelucci[1, 6] also reported a series of observations which were in favor of a reflex migration of the pigment in the frog's retina. He found that this migration could be produced in both eyes by pressure on one eye or by mechanical irritation of the skin of the animal. He even contended that sound waves were able to produce the same effect. Herzog[7] had similar results with mechanical stimulation and paid special attention to the influence of the temperature on the phe-

* From The Dartmouth Eye Institute, Dartmouth Medical School. Read before the Association for Research in Ophthalmology, at San Francisco, June 14, 1938.

nomenon. This had already been studied by Gradenigo[8] and was later carefully investigated by Arey[9] and Detwiler and Lewis.[10] It has been shown conclusively that frogs, kept at certain temperatures, for a sufficient time, in complete darkness, show marked light position of their retinal pigment. The same result in fishes has been reported by Wunder.[11]

One fact mentioned in Herzog's paper cited above is of special interest. Herzog stated that the retinal cones were unusually long in decerebrated frogs and in frogs in which the spinal cord was destroyed. This he ascribed to the lack of central tonus. Dittler[12] could not confirm this finding, but Garten[13] found that it was true for animals in which he had severed the optic nerve. Hamburger[14] saw this phenomenon in opticotomized frogs only occasionally.

Although all these results seem rather convincing, serious objections have been voiced against their conclusiveness. Fick[15] in 1889 already pointed out that in Engelmann's experiments in which only one eye was exposed to light vestiges of light might have reached the other eye. In the experiments in which the head of the animal was covered with a light-tight cap and the skin of the body exposed to light, the respiration might have been impeded, creating a change in the constitution of the blood. This might act as a humoral stimulus on the retinal epithelial cells. Finally, it is possible that in the various parts of the retina there are differences as to the migration of the pigment.

Much more serious obstacles for the assumption of a nervous regulation of the pigment migration in the frog's retina arose, however, when Dittler[12] could show on isolated retinas that a full contraction of retinal cones takes place under the influence of acids. His experiments on the isolated retina of the frog kept in Ringer's solution showed that the exposure to light causes the formation of acid products and that these products are able to provoke a maximal reaction of the cones. Similar results had been reported by Lodato,[16] before those of Dittler were published. He had found by other methods that there is a close relation between the chemical reaction of the retina and the changes that occur in the layer of the rods and cones. The expansion of the cones corresponds to an acid reaction of the retina, their contraction to an alkaline reaction. Retinas of light-adapted frogs, therefore, always have a marked acid reaction, retinas of dark-adapted frogs show an alkaline reaction, rarely a neutral, and very rarely a slight acid reaction. The injection of strychnine produces an acid reaction of the retina. The same is true for faradization of the animal and, though to a lesser degree, for an elevated temperature (35°-40°C.), acting on the animal for from three-quarters of an hour to one hour.

In the light of these results it seemed quite justifiable to question, whether all the aforementioned reflex movements of the retinal pigment are really to be considered as such, and, up to this time, the current opinion expressed in all textbooks (see, for example, Kolmer and Lauber[17]) that treat of this question is that so far nothing has been found which would be definitely in favor of an influence of the central nervous system on the pigment epithelium of the retina.

Experimental investigations and results

From this short review of the literature it appears that the question as to whether and to what extent there is a regulatory influence of the central nervous system on the pigment migration in the frog's retina, is quite unsettled. The contribution to this particular problem was motivated by the results of previous experi-

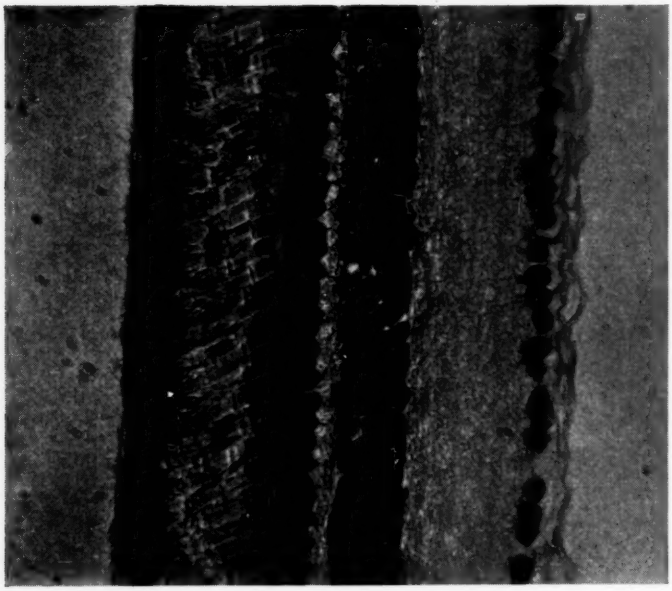


Fig. 1 (Burian). Retina of normal dark-adapted frog (control animal no. 12). The bulk of the pigment is located in the body of the pigment cells. ×400.

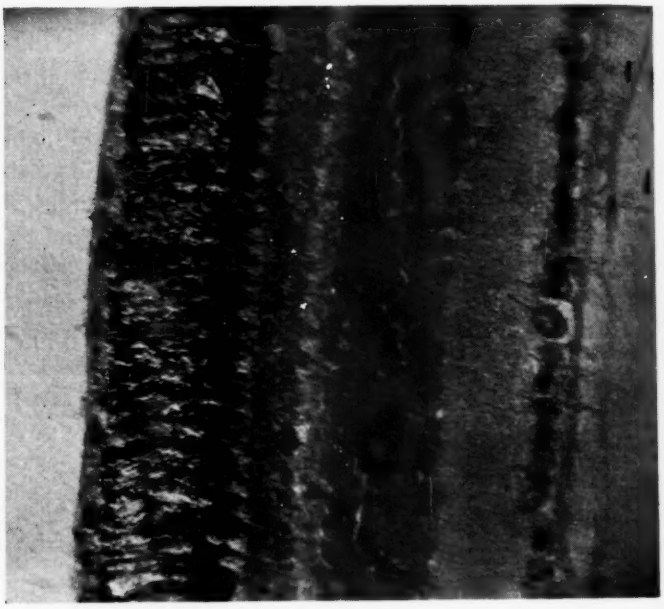


Fig. 2 (Burian). Retina of dark-adapted frog poisoned with strychnine (animal no. 13). The pigment presents maximal migration. Virtually all pigment is accumulated at the external limiting membrane. ×400.

ments,[18] concerning the influence of naphthalene on the spinal cord of the frog.

The experiments to which we refer showed that an appropriate dose of naphthalene administered to a frog, is able to produce a typical poisoning of the frog. At the height of the poisoning, the frog shows absolute muscular atony and a complete lack of mechanical reflex response, while the chemical, thermal, and electrical reflex excitability remain perfectly normal. Exhaustive quantitative studies of this quite unusual reflex phe-

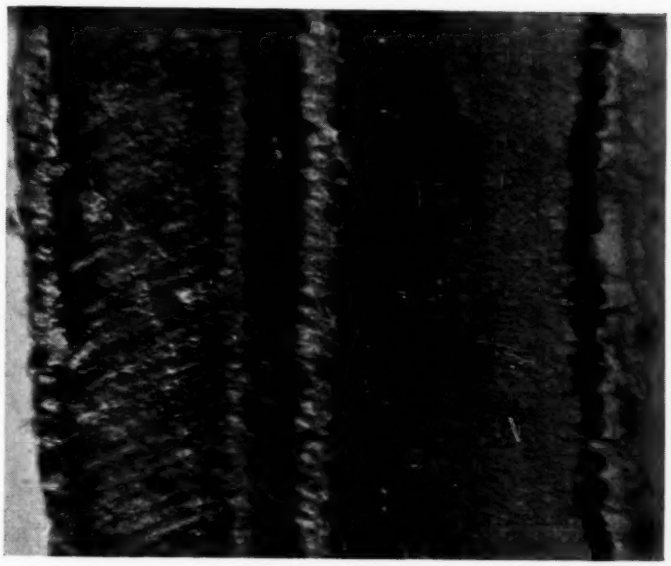


Fig. 3 (Burian). Retina of normal light-adapted frog (control animal no. 15). Most of the pigment is scattered along the rods and cones. ×400.

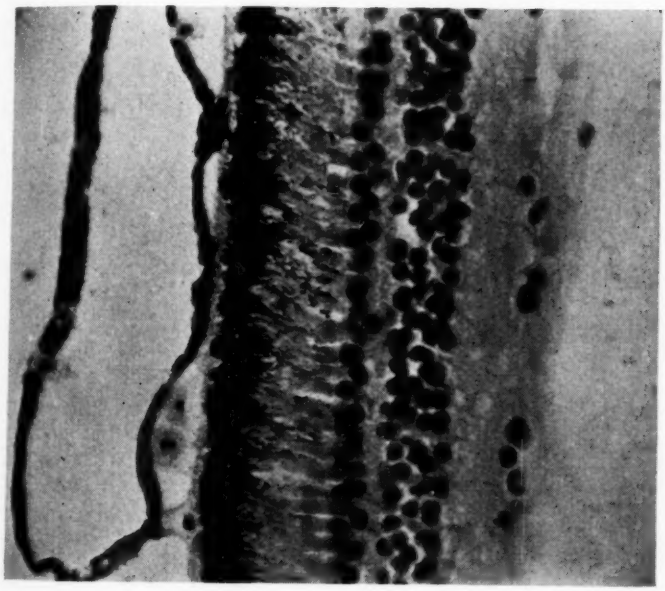


Fig. 4 (Burian). Retina of light-adapted frog poisoned with naphthalene (animal no. 16). The pigment is concentrated in the bodies of the cells. ×400.

nomenon and the combination of naphthalene poisoning with strychnine or phenol poisoning gave results which can be summed up briefly as follows:

Naphthalene affects the center, not the periphery of the neuro-muscular apparatus. The action of the poison is directed against the sensory, not the motor part of the spinal cord, and the seat of the poisoning is to be sought in the so-called intercalary neurones. Strychnine acts as a perfect antagonist to naphtha-

lene. While strychnine raises to the highest level the readiness of the central nervous system to react to mechanical stimuli, producing the well-known strychnine tetanus, naphthalene completely abolishes it. Consequently, the central tonus of the muscles is maximal in strychnine poisoning and virtually equal to zero in naphthalene poisoning. I shall not enter into the discussion of the mechanism of the strychnine and naphthalene poisoning, the study of which throws an interesting light on the functional structure of the spinal cord, but would state that the antagonism of strychnine and naphthalene extends also to the chromatophores of the skin. While a frog poisoned with strychnine is extremely light in color, due to the maximal contraction of the chromatophores, a frog intoxicated with naphthalene is almost invariably very dark, due to the full expansion of the chromatophores. This reaction of the chromatophores is another sign of the extremely high or extremely reduced central tonus in strychnine and naphthalene poisoning.

The behavior of the chromatophores induced me to investigate whether or not the two drugs had a similar antagonistic influence on the position of the retinal pigment. As mentioned before, Engelmann had already found that dark-adapted frogs poisoned with strychnine showed light position of the pigment and contraction of the cones. Hess[19] on the other hand, reports in one of his papers that he repeatedly observed, at various places in the pigment epithelium of rabbits intoxicated with naphthalene, large groups of cells arranged in the form of a circle around a center in which all the pigment was located at one border of the individual cells, either at the border turned toward the center of the circle or at the border turned away from that center.

I conducted four series of experiments, each consisting of a number of individual tests. In the first series I repeated Engelmann's experiment with strychnine and invariably found that thoroughly dark-adapted frogs showed maximal expansion of the pigment—that is, marked light position—when poisoned with strychnine. Figure 1 shows the histologic section of the retina of a dark-adapted control animal, and figure 2 the section of a dark-adapted animal poisoned with strychnine.

In a second series, light-adapted frogs were poisoned with naphthalene. The retinas of these animals always showed a marked dark position of the pigment; that is, the pigment of the cells was accumulated in the body of the cell while the cellular processes were entirely free of pigment. This, together with the appearance of the retina of a light-adapted control animal, is shown in figures 3 and 4.

These two series of experiments seemed rather convincing, indicating that the position of the pigment could be controlled by influencing the central nervous system.

In order to confirm this assumption and to form an idea of the pathway by which this influence is exerted, performed two further series of experiments. In both series the optic nerve of one side—always the left one—was severed. In frogs this is done very easily through the roof of the mouth. The animals were then submitted to dark or light adaptation and poisoned either with strychnine or naphthalene. The result was unequivocal. The retina of the normal eye always showed the position which it had assumed under the influence of the poison in the animals not operated upon; however, the fellow-eye, which had been operated upon, presented a more or less intermediate position. In light-adapted frogs there was some indication of expansion of the pigment; in dark-adapted frogs the pigment was accu-

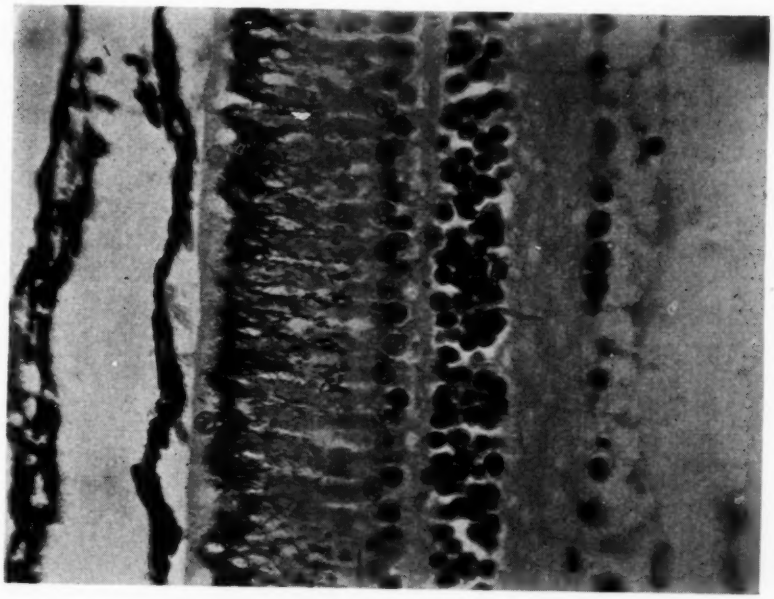


Fig. 5 (Burian). Normal frog, light adapted. Retina of the eye, the optic nerve of which was severed seven days previous to experiment (control animal no. 14). Most of the pigment in the cell bodies, some pigment scattered along the rods and cones. ×400.

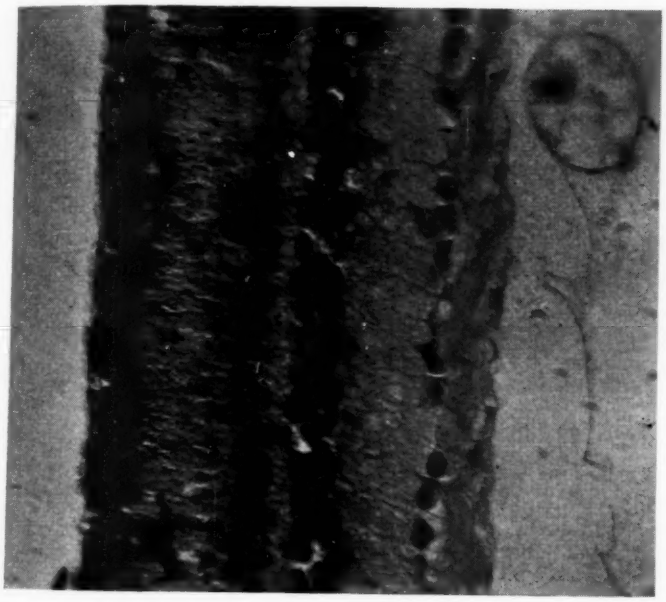


Fig. 6 (Burian). Dark-adapted frog, poisoned with strychnine (animal no. 19). Retina of eye in which opticus had been severed 18 days previous to experiment. Essentially the same distribution of pigment as in figure 5. ×400.

mulated more in the body of the cells. Figures 5, 6, and 7 clearly show this behavior.

As to the *technique* of the experiments, the following may be said:

All animals were kept under exactly the same conditions. The dark adaptation was accomplished by keeping the animals in a completely darkened room for from 12 to 24 hours. As a further precautionary measure, the jars containing both the experimental and the control animal were covered with a thick black cloth. The strychnine poisoning was done by injecting a measured amount of 0.1 percent

solution of strychnine nitrate into the dorsal lymph sac of the animal. The injection was performed in complete darkness; the enucleation at the height of the strychnine tetanus, with the aid of a neon light to which the control animal was equally exposed. The light-adapted frogs were kept for from 6 to 10 hours in a very light room; the administration of naphthalene was made orally, with the aid of a syringe on which a small rubber tube was mounted. The naphthalene used was an oily solution in the proportion of one part naphthalene to eight parts of paraffin oil. Various fixatives were tried out; the best results were obtained with Held's solution modified by Kolmer,[20] and we followed the technique suggested by Detwiler,[21] fixing the bulbi for 12 hours and afterwards placing them in running water for 12 hours. The eyes were then opened at the equator and small circular pieces punched out of the posterior part of the bulbus with a trephine, which always allowed the use of analogous parts of the retinas of the various animals. The choroid and sclera, remaining in contact with the retina, made the further treatment of the specimens easier than if only the retinas had been used. The specimens were embedded in paraffin; their orientation in the block was such that the direction of movement of the microtome blade was parallel to the rods and cones. In this way they were not pushed together nor crushed. The sections were stained with hematoxylin and eosine.

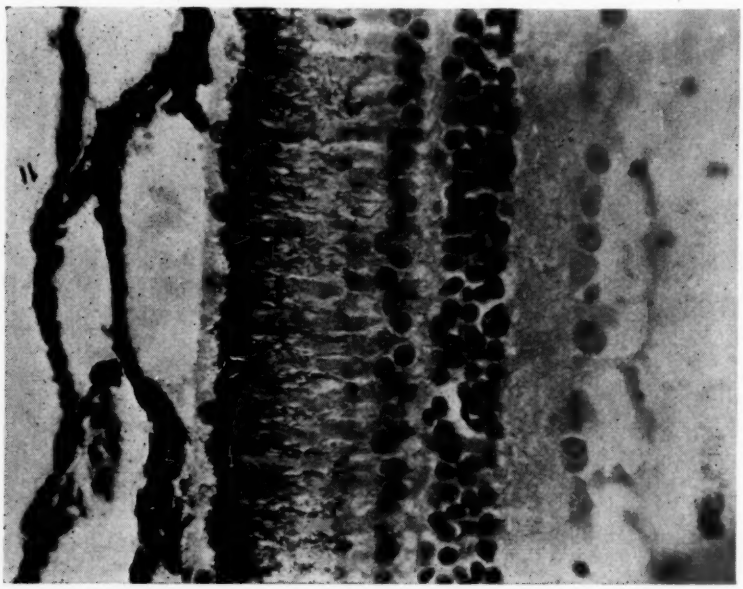


Fig. 7 (Burian). Light-adapted frog, poisoned with naphthalene (animal no. 20). Retina of eye in which the optic nerve had been severed 18 days previous to experiment. Essentially same distribution of pigment as in figures 5 and 6. ×400.

Conclusions

It seems to me that the results of the experiments permit only one explanation. It has been shown clearly by pharmacological experiments that the effect of both strychnine and naphthalene is a central one; that is, that the manifestations of poisoning which were observed were due to an affection of the central nervous system. Since I was able to influence the position of the retinal pigment by the administration of these drugs, it may be assumed that this position was influenced by way of the central nervous system. In other words, the position of the retinal pigment is influenced by a central nerv-

ous tonus, as had been previously assumed by Herzog, and in the present experiments it was possible to change the position of the retinal pigment by raising or lowering this tonus. If the central tonus is brought to a maximum artificially, as is the case in strychnine poisoning, the pigment assumes a light position and is expanded even beyond the normal limit. In keeping with the accepted explanation of the effect of strychnine, it may be assumed that the excitability of the sensory part of the central nervous system has become maximal and that, therefore, stimuli of relatively low intensity lead to a maximal effect. A reflex migration of the retinal pigment, such as Angelucci found in his experiments with mechanical stimulation of the skin, would, under these conditions, be extremely favored.

The opposite is true for naphthalene. These experiments have shown that naphthalene lowers the central tonus of the muscles to a minimum, due to the reduction in the influx of impulses into the central nervous system. The sensorial part is, so to speak, blocked. According to the lack, or extremely low level, of the central tonus, light-adapted naphthalene frogs show unexpanded pigment.

The experiments of Lodato and Dittler, cited in the introduction, do not seem to invalidate my statements. It has been proved beyond doubt, that light as well as other factors (strychnine poisoning, heat, faradization) produce an acid reaction of the retina and that acids in their turn are able to provoke a contraction of the cones. Hence, it may be concluded that the local condition that gives rise to the pigment migration is the formation of acids. These acids, however, can be produced by various means: by direct illumination, by changes due to nervous influences, and, finally, by local and general humoral changes.

The influence of light as well as of the general humoral conditions is not restricted to the retina. Light, as is well known, has a definite effect on the central nervous system, affecting the level of its basic tonus, which is, of course, also under the influence of the humoral conditions present in the body.

In the light of these conclusions we should consider all experiments concerning the pigment migration induced by other stimuli than light—that is, the various reflex movements—reported in the introduction, and the movements due to various drugs (cocaine, santonine, adrenalin).

Finally, as to the pathway by which the central nervous system exerts its influence, it seems to me, in accordance with the reported experiments in which the optic nerve had been severed, that the old conception of Engelmann is justified, that the optic nerve contains centrifugal, retinomotor fibers. This conclusion is contrary to that at which Shoko Kyo, a Japanese author, arrived on the basis of his experiments, which are reviewed in the Zentralblatt für die gesamte Ophthalmologie.[22] He states that in frogs in which the optic nerve had been severed unilaterally, faradization as well as the application of drugs which produce a migration of the retinal pigment have the same retinomotor effect on both eyes. He does not believe, therefore, that the retinomotor impulses are transmitted through the optic nerve. I am unable to say to what this difference in the findings is due, all the more since Kyo's paper was accessible to me only in the review mentioned. In another paper Kyo[23] reports the results of experiments in which he has studied the effects of a great number of drugs on the pigment migration of the frog's retina. His conclusion is that drugs, stimulating the central nervous and the sympathetic nervous system, cause a migration of the retinal pigment, while

drugs having the opposite effect on the nervous system do not influence the retinal pigment. This, according to my experience, does not apply to naphthalene.

The author is greatly indebted to Dr. Ralph E. Miller of the Department of Pathology, for his generous help. The histological sections were excellently done by a technician on the staff of Dr. Miller's laboratory. Dr. Adrian Kameraad, of the Department of Biology, was kind enough to make the microphotographs reproduced in this paper.

Summary

Experiments with strychnine and naphthalene have shown that the retinal pigment of dark-adapted frogs shows light position when the animals are poisoned with strychnine, and that in light-adapted frogs poisoned with naphthalene the retinal pigment assumes dark position.

From this the author concludes that there is a regulating influence of the central nervous system on the position of the retinal pigment.

In frogs, in which the optic nerve on one side was severed, the drugs did not influence the eye operated upon. The author concludes that the pathway through which the central nervous system exerts its influence must be located in the optic nerves, supporting Engelmann's theory of the existence of centrifugal, retinomotor fibers in the optic nerve.

The position of retinal pigment appears to be influenced by the following factors: (1) the basic tonus of the central nervous system; (2) the "light tonus" of the central nervous system; and (3) local humoral conditions due to the influence of light and various general metabolic factors.

Bibliography

[1] Angelucci. Arch. Anat. u. Physiol., 1878, p. 353.
[2] Boll. Ibid., 1877.
[3] Kühne. Heidelb. Unters. 1878, v. 1.
[4] Engelmann. Pflüger's Arch., 1885, v. 35, p. 498.
[5] Van Genderen Stort. Graefe's Arch., 1887, v. 33.
[6] Angelucci. Encyclopedie franç., 1905, v. 2.
[7] Herzog. Arch. f. Anat. u. Physiol., Physiol. Abt., 1905, p. 413.
[8] Gradenigo. Allg. Wien. med. Zeitg., 1885, v. 30, p. 343.
[9] Arey. Jour. Comp. Neur., 1916, v. 26, p. 126.
[10] Detwiler and Lewis. Jour. Comp. Neur., 1926, v. 41, p. 153.
[11] Wunder. Zeit. vergl. Physiol., 1925, v. 3, p. 1.
[12] Dittler. Pflüger's Arch., 1907, v. 117, p. 295.
[13] Garten. Graefe-Saemisch Handbuch, Ed. 2, 1907/1908, v. 3, chap. 12.
[14] Hamburger. Quoted by Garten.[13]
[15] Fick. Graefe's Arch., 1891, v. 37.
[16] Lodato. Zeit. f. prakt. Augenh., 1900, v. 26, p. 380.
[17] Kolmer and Lauber. In Handb. d. mikrosk. Anat. d. Menschen, 1936, v. 3, pt. 2.
[18] Burian. Pflüger's Arch., 1937, v. 238, p. 651.
[19] Hess. Heidelb. Ber., 1902, v. 30, p. 336.
[20] Kolmer. Pflüger's Arch., 1909, v. 129, p. 35.
[21] Detwiler. Jour. Comp. Neur., 1923, v. 36, p. 125.
[22] Kyo. Zent. f. d. ges. Ophth., 1938, v. 39, p. 645.
[23] ———. Zent. f. d. ges. Ophth., 1938, v. 40, p. 427.

Discussion

Dr. Frederick H. Verhoeff (Boston): I should like to ask the essayist whether or not he has excluded the possibility that cutting the optic nerve has affected the retina in such a way that it will not respond. If you cut the optic

nerve, it affects the retina profoundly, for subsequently the ganglion cells disappear. I have always assumed that the effect was very great the moment you injured the optic nerve; that it was due to changes that take place, electrical and chemical, the result of the cutting, and that these changes cause impulses to pass backward along the nerve and injure the ganglion cells.

If the retina has been injured, of course, you would not expect it to respond to the action of a drug as does a normal retina.

Assuming, however, that this is not so, is it necessary for the essayist to assume that there are special nerve fibers going back into the retina?

We know if you stimulate any nerve, motor or sensory, impulses will pass in both directions. I think it was Head who drew attention to what he called antidromic impulses that pass backward along sensory nerves. Isn't it possible that in the optic nerve the fibers that ordinarily carry the impulses to the brain would serve the purpose for which the essayist hypotheticates special nerve fibers?

Then I should like to have Dr. Burian make a diagram illustrating his conception as to how nerve impulses passing to the retina affect the pigment cells. He may have several different ways to suggest.

DR. BURIAN: The first point brought out by Dr. Verhoeff, of course occurred to me, too. It is quite probable that the cutting of the optic nerve profoundly affects the eye; however, I thought that this, at least to a certain extent, could be paralyzed by taking the animals in various stages after the cutting of the optic nerve, or up to 20 days after the cutting of the optic nerve.

As to the retinal motor fibers, whether there really are retinal motor fibers or whether it is a question of conduction of the impulse both ways, I could not say; for the experiments on which I am reporting do not explain this sufficiently. However, in view of Engelmann's experiments, which show that there is not only a direct influence from the brain to the eye, but from one eye to the other, we have necessarily to assume that there must be some kind of reflex arc and, therefore, afferent and efferent fibers and a center should be present.

DR. VERHOEFF: It doesn't seem to me that that follows. All that is necessary is for impulses to get back to the other eye.

DR. BURIAN: I can't quite see how the impulses would come from one eye through the brain, through the centers to the other eye, without having a special afferent fiber.

DR. VERHOEFF: It seems to me it would be very simple. Somewhere fibers from the two eyes go to the same place; otherwise an image from one retina could not be unified with one from the other retina. Hence impulses from one retina could be carried back to the other.

DR. BURIAN: My talk was not concerned especially with the retinal motor fibers. What I wanted to show most was that there was an influence of the central nervous system on the pigment migration. The way it is carried on is, I thought, by special fibers—or else by the same fibers, only with other impulses going the other way, and my experiments, unfortunately, don't give any answer to that.

DR. VERHOEFF: I take it that now you admit that your investigation does not prove the existence of special fibers.

DR. BURIAN: I still think as we usually do, that when there is a reflex action, there must be three elements.

DR. VERHOEFF: What we usually think isn't necessarily true.

DR. BURIAN: I didn't want to go into a criticism of the reflex action.

DR. VERHOEFF: Well, you brought it up!

DR. BURIAN: Yes, I did, but I don't think it is quite that way. I feel that the explanation that there are special fibers, is simpler than to assume that in the same nerve fiber the impulses go back and forth.

DR. VERHOEFF: It is more complicated to think there are two things, than one.

DR. BURIAN: Not necessarily, I think.

DR. VERHOEFF: It is to my mind.

DR. BURIAN: The third question I do not quite understand.

DR. VERHOEFF: You claim that there are these special fibers. I want you to draw a diagram of a retina and show how they act.

DR. BURIAN: I wouldn't be able to make a diagrammatic drawing of the effect of the impulses on the retinal pigment.

DR. VERHOEFF: Roughly explain how you think the impulses act.

DR. BURIAN: The way by which they act?

DR. VERHOEFF: You say that the impulses come to the retina and affect the pigment epithelium. We would like to have some idea just how they could do this. You must have some conception as to this because you think it possible. Just mention one way you think it could happen.

DR. BURIAN: I don't have any special conception about that, about the mechanism of this effect; you mean, anatomically?

DR. VERHOEFF: You have that nerve impulse coming to the retina. I want to know what it does after it gets there.

DR. BURIAN: I have no special—

DR. VERHOEFF: I think it is rather difficult to assume the impulses if you can't think of any way they can act.

DR. BURIAN: There are probably various ways, but I brought out modestly the fact that this occurs. I haven't attempted any explanation.

DR. VERHOEFF: That leaves it a little doubtful that nerve impulses are concerned in the matter. Perhaps the effects are produced directly from the blood.

DR. BURIAN: I think the important point is that it is not produced by the blood. We know from other pharmacological experiments that these two drugs, strychnine and naphthalene, affect a definite part of the central nervous system. There is no reason why suddenly, in this case, these drugs should affect by way of the blood.

DR. VERHOEFF (interrupting): You don't mean to say that the retina isn't part of the nervous system, do you?

DR. BURIAN: Oh, yes, that is right.

DR. VERHOEFF: Why couldn't that be affected directly?

DR. BURIAN: It still wouldn't be by way of the blood. You see, the nerve endings are also part of the nervous system, and are not directly affected by strychnine and naphthalene.

DR. VERHOEFF: You are sure they are not?

DR. BURIAN: In the case of naphthalene—

DR. VERHOEFF (interrupting): In the retina, you mean?

DR. BURIAN: I wouldn't have any way of checking that in the retina. The influence of strychnine, we know, in man is not peripheral—or do you think so?

DR. VERHOEFF: I don't know, as concerns the retina. You are the one trying to tell us that.

DR. BURIAN: Not in man.

DR. VERHOEFF: I want to know how conclusive your evidence is.

DR. BURIAN: I don't know whether there is anything else.

DR. VERHOEFF: I haven't anything else in mind.

DR. BURIAN: Thank you.

A CONTACT-LENS–TELESCOPIC SYSTEM*

JEROME W. BETTMAN, M.D., AND G. STUART MCNAIR
San Francisco

Attempts have been made for centuries by the use of innumerable optical devices to help those unfortunate individuals who have suffered a drastic and permanent reduction of visual acuity. In 1646, Kircher designed a hand telescopic spectacle for near use. Dixon, in 1785, constructed a telescopic spectacle from two spherical mirrors, which were later replaced by spherical lenses. In more recent times, Hertel and Von Rohr developed the familiar Zeiss telescopic spectacle. The Hamblen Company of England and Feinbloom of New York likewise devised telescopic spectacles. The Feinbloom type is designed on the anomophotic principle: magnification in the horizontal meridian is ×1.8, while in the vertical meridian it is only ×1.3. With this type an image magnified ×1.8 is seen at a distance which seems to approach more nearly the actual distance.

The ordinary types of telescopic spectacles are large, heavy, and conspicuous. The patient's field of vision is restricted to a dangerous degree. Many ophthalmologists have reported that most of their patients who can secure improvement in visual acuity in no other way cannot be persuaded to wear the telescopic lenses in public. Even the nearly blind are vain about their personal appearance and the restricted visual field is both an annoyance and a hazard.[1]

To overcome these difficulties we conceived the idea of producing a telescopic spectacle by using a contact lens for the eyepiece and a highly convex ophthalmic lens for the objective. The appearance of such an arrangement is only slightly more objectionable than that of the ordinary cataract lens. The field of vision is larger than with the ordinary telescopic spectacle of ×1.8 magnification, with which the visual field is reduced to 24 degrees.

A search of the available literature revealed the fact that the theoretical possibilities of this arrangement had been suggested by Dallos[2] and Boeder;[3] however, neither of them tested the principle in actual practice.

Before proceeding with the description of this telescope, let us briefly consider some of the properties of telescopic spectacles in general. All telescopic spectacles are constructed on the Galilean principle, a positive objective and a negative eyepiece separated by the difference of their focal lengths. This practice is followed because:[4]

1. Galileo's is the only system in which two simple lenses produce an erect image. Other telescopic systems need an erecting system which makes them thicker and heavier and results in additional loss of light (4 percent at each glass-air surface).
2. In other telescopic systems the lenses are separated by the arithmetic sum of their focal lengths, not their difference; therefore, the telescope must be longer.
3. The Galilean type of telescope produces less astigmatism with a large flat field than do other types of telescopes.
4. Galilean telescopes have a large exit pupil.

The following simple formulas govern the construction of a Galilean telescope:

$$\frac{F1}{F2} = \text{magnification}$$

Where F1 = focal length of the objective lens, F2 = focal length of the eyepiece.

The lenses must be separated by the difference between their focal lengths, that is, F1 − F2 = D.

The following simple principles must

* From the Department of Ophthalmology, Stanford University Medical School.

be followed in the construction of a telescopic spectacle:[5]

1. The telescope must be of relatively low magnification, otherwise the speed of movement of objects and the reduction of visual field is not tolerable.
2. The interpupillary distance of the telescopic spectacle must equal that of the wearer. If it does not, the symmetry of the visual field and the definition of the image are impaired.
3. The frame must be absolutely rigid so that tilting of the spectacle around the anteroposterior axis cannot occur. When a telescopic spectacle is tilted, the periphery of the optical system is brought into use. This results in increased chromatic aberration and astigmatism.

The patient for whom we devised this contact-lens–telescopic system was unable to wear an ordinary telescopic spectacle. The unsightliness of the telescopic spectacles was a grave handicap in his search for employment even though his vision was improved from 8/200 to 20/100 +1 by them.

As far as we know the only type of telescopic lens previously designed to overcome the impairment of visual field and the unsightliness of the usual telescopic spectacle is that advocated by Berliner.[6] The lens that Berliner described was designed by Polackoff and developed by the Univis Company. It consisted of strong positive and strong negative flint lenses of a high index of refraction separated by a Crown glass component of a low index of refraction—all components being permanently fused together. This solid-glass telescope was then set into a carrier lens mounted in an ordinary spectacle frame. The Univis type is definitely an advance over the customary type of telescope, but is certainly not so inconspicuous as one would desire.

The contact-lens–telescopic system here described was designed to be as inspicuous as possible and to allow the patient to use his entire visual field. A contact lens served as the eyepiece, and a biconvex lens in a spectacle frame was used as the objective. To avoid undue separation between the spectacle (objective) and contact lens (eyepiece), it was apparent that the effective diverging power of the contact lens must be as great as possible. Only then could an objective (spectacle lens) of high power and short focal length be used, so that both the desired magnification and the short distance between the objective and eyepiece would be obtained.

A few examples will serve to emphasize this: A simple telescope with a magnification of ×2 was constructed by Eggers[7] by using a +5.00 diopter objective and a −10.00 diopter eyepiece separated by the difference of their focal lengths, 10 cm. If a +10.00 D. objective and a −20.00 D. eyepiece had been used, the separation would have been only 5 cm.

The cornea acts as a converging lens of approximately 45 diopters because of its convex curvature. A contact lens eliminates the cornea as a refracting medium. The converging power of the contact lens, which depends upon the curvature of the corneal segment of the contact lens, is substituted for that of the cornea. Consequently, we decided to use a contact glass on which an entirely flat surface had been ground, as this would optically eliminate the cornea as a converging lens. In other words, the cornea, which acts as a strong converging lens, could be optically neutralized. Therefore, the same effect would be produced as when a strong minus lens of a dioptric power equal to that of the cornea is held in front of the eye. Our patient's corneal curvature was found to be 44 diopters with the keratometer; consequently, with this eliminated by the flat-surface contact lens, the effect produced would be that of a −44 diopter lens. This, then, would serve as the minus lens (eyepiece) of a Galilean telescope. One would then expect

a magnification of ×2 with an ordinary + 22 diopter lens (objective of the telescope) set in a spectacle frame 27 mm. from the eye (the difference of the focal lengths of the two lenses). To reduce the cosmetic disfigurement, a stronger plus lens, a +29 biconvex lenticular, was used at a shorter distance from the contact lens (18.5 mm.) with approximately ×1.6 magnification. With this combination of lenses, the patient was able to read 20/100.

The contact glass was ground on the principle of the Dallos contact glass.[8] In this type of glass the optically effective region of the corneal portion is only the central 8 mm. This permits the grinding of a much higher correction on the contact glass without excessive increase in weight or thickness of the glass. In our contact glass the optically effective portion was restricted to 6 mm., on the anterior surface of whose corneal segment a flat surface was ground. The posterior surface of the corneal segment was made sufficiently concave just to clear the cornea, as is the case with an ordinary contact lens. The scleral curve was that which fit with the greatest comfort (again as with the ordinary type of contact lens). The optically effective portion was restricted to 6 mm. instead of 8 mm., as in the Dallos contact glass, in order to facilitate grinding without increasing the weight of the glass. Observation of the patient's pupil showed that it did not dilate over 4 mm. under ordinary conditions; therefore, a 6-mm. corrected corneal portion was sufficient. The weight of the finished contact lens is 0.6 gm. instead of the usual 0.5 gm., is thoroughly comfortable, and has been very satisfactory.

The objective lens for distance was a +29.00 diopter lenticular type, biconvex lens set in a very rigid frame made of zylonite and metal. A metal bridge and pad arms were used to obtain an exact adjustment and rigid fit. The pad arms were especially made from solid gold stock, tempered to the proper hardness. This was necessary because of their unusual length of 19 mm.

As mentioned above, the patient is able to read 20/100 with this contact-lens–telescopic system, without experiencing vertigo or excessive chromatic aberration. A plano balance was placed in the spectacle frame before the uncorrected eye so that the patient might have the advantage of a large field (vision in this eye was 6/200). No diplopia nor confusion was caused by leaving this eye open. No attempt was made to correct the poorer eye with the same type of telescope.

In the Zeiss telescopic spectacle the reading addition is placed on the front of the object lens. The standard reading addition is a +4 lens. The magnification obtained with this reading addition is ×1.8, the same as that obtained with the distance portion. A +8.00 D. reading addition gives twice the magnification of the distance portion, 2 times 1.8, or ×3.6. To read newspaper print fairly well without further magnification in the reading addition the patient's distance vision with the telescopic lens should be about 20/50. If it is less than this the reading addition should be greater than a 4-diopter lens so that further magnification is obtained.[9] Thus, a patient with 20/100 vision with a distance telescope should have a +6.00 D. reading addition. This is done to supply a magnification of ×1.5 for reading besides the ×1.8 magnification that he already has for distance. Thus, a total magnification of ×1.5, 1.8, or 2.7 is available for reading. The reading distance, depth of focus, and visual field are reduced with further increase in the magnification; therefore, additional magnification for reading should not be prescribed unless it is necessary.

The visual acuity of our patient was

20/100 with this telescopic system. Accordingly, we decided to use a +6.00 D. addition for reading. A +35.00 D. biconvex lenticular lens, incorporating the +6.00 D. addition with the +29.00 D. objective, was mounted in another spectacle frame similar to the one described above. The finished spectacle is shown in figure 1.

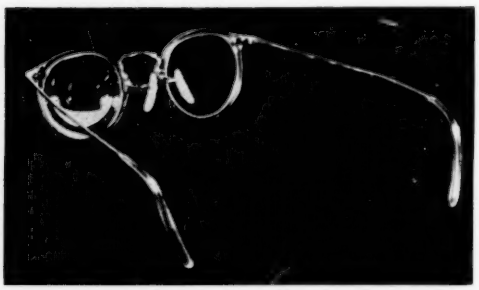


Fig. 1 (Bettman and McNair). Spectacle of the contact-lens–telescopic system showing long pad arms.

With this the patient could read Jaeger 1 with some difficulty at a distance of 13.5 cm. He was able to read 48 words of ordinary size newspaper print a minute. With his Zeiss telescopic spectacle and +8.00 D. reading addition he was able to read only Jaeger 2 and 35 words per minute. The cosmetic improvement can be seen by comparing figures 3 and 4.

For those who may wish to prescribe the contact-lens-telescopic spectacle described, the following outline of procedure may be of value:

1. Determine the refractive error.
2. Measure the corneal curvature with a keratometer.
3. Try on contact lenses of the ordinary type from a trial set to determine the correct scleral curvature and to be certain of the clearance of the corneal segment. The radius of curvature of the cornea may also be determined from Obrig's table.[10]
4. A contact lens of the Dallos type with a 6-mm. flat anterior surface and the posterior corneal and scleral radii determined in step 3 may then be ordered. Should the patient's pupil be larger than 4 mm., the flat surface of the contact lens may have to be somewhat larger.
5. A convex lens of approximately one half the dioptric power of the corneal curvature (which has been optically eliminated by the contact lens) should be placed before the eye with the contact lens in place. The distance between the contact lens and the objective lens must be approximately the difference between the focal lengths of the objective lens and the eyepiece (contact lens). The optimum distance between the objective and eyepiece and the power of the objective are somewhat modified by the magnification desired, the refractive effect of the fluid meniscus between the contact lens and cornea, and any existing spherical ammetropia of the eyeball.[9] The details can be worked out with a trial set.
6. A biconvex, preferably lenticular, lens is ordered and placed in a very rigid frame. The nose pads are built up to hold the lens at the required distance from the contact lens.
7. The optical center must be adjusted to coincide precisely with the center of the patient's pupil. The slightest variation will blur the image.
8. An objective lens for reading equal to the power of the distance objective lens plus the reading addition required is then mounted in a frame similar to the one used for the distance objective lens. A 4.00 D. addition is sufficient if no magnification other than that given by the distance telescope is needed (that is, if the distance vision is 0.5 or better with the telescope). Stronger reading additions to produce greater magnification may be used as required.

It is wise to test the patient with a telescopic lens of the usual type from a trial set, to be certain that substantial improvement in vision will be obtained, before he is subjected to the expense and time necessary to obtain a contact-lens-telescope.

Case history. J. G., a white male, aged 50 years, was first seen in October, 1936. He had had no difficulties until two years before entry, at which time his vision failed gradually. His general health had always been excellent.

Examination: Vision was R.E. 7/200; L.E. 8/200; but no Jaeger for either eye. Both eyes were normal externally.

Fundi: White raised areas of fibrous-like tissue in the macular regions were seen in both eyes. That in the right eye was: 1 by 1½ disc diameters in size (fig. 2); that in the left eye, 2½ by 4 disc diameters. One small superficial hemorrhage was present at the edge of the area in the right eye. The fundi were otherwise normal. The tension also was normal, as were the ocular rotations.

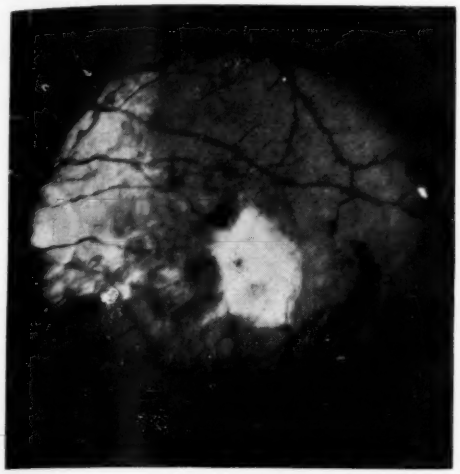


Fig. 2 (Bettman and McNair). Photograph of fundus of patient's left eye showing raised, white, tumorlike mass in macular area. This eye was corrected to vision of 20/100+ and Jaeger 1 with difficulty by using the contact-lens–telescopic system.

Retinoscopy: R.E. +.25 D. cyl. ax. 180°; L.E. +.50 D. sph. ⊂⊃ +.50 cyl. ax. 120°

Visual fields: Absolute central scotomata were present in each eye with a 1.5-mm. white object on the Walker screen at one meter. The scotomata extended irregularly between the 5° and 20° circles in each eye. The peripheral fields were normal.

The general medical examination uncovered no pathology.

Laboratory examination: Blood, urine, blood Wassermann, and spinal fluid were negative.

Impression: Disciform macular degeneration.[11]

Telescopic lenses of the Zeiss type were fitted from a trial case. The following lenses resulted in the vision indicated:

O.D. 0 Base Zeiss × 1.8 with −50. D. sph. 20/200

O.S. 0 Base Zeiss × 1.8 with −50. D. cyl. ax. 60° 20/100 +1

Add O.D. +8.00 —Jaeger 16
O.S. +8.00 —Jaeger 3

(The above was prescribed for the left eye only.)

In August, 1937, the patient returned having the same vision with the telescopic spectacles as when they were prescribed. He had been able to use the lenses successfully. However, the unsightly appearance of the spectacles and his inability to see without them had caused his application for several jobs to be rejected. The reduction in visual field was annoying to him. Accordingly, the procedure outlined above was carried out and the following contact-lens–telescopic spectacle was prescribed for the left eye.

Eyepiece: 2/7.5 Dallos-type contact lens with flat anterior corneal surface 6 mm. in diameter.

Objective lenses: Biconvex lenticular, +29.00 D. sph. for distance, +35.00 D. sph. for reading.

The *mounting* consisted of a special rigid metal frame with nose pads built up.

With this combination the patient was able to see 20/100+ in the distance and could read Jaeger 1 with some difficulty. His visual field for distance was no longer restricted. The lens looked far less conspicuous than the usual telescopic lens, although not so inconspicuous as an ordinary spectacle. The patient experienced no untoward symptoms such as color aberration, vertigo, or headaches. He has but little difficulty in keeping his place

Fig. 3 (Bettman and McNair). Patient wearing Zeiss telescopic spectacles.
Fig. 4 (Bettman and McNair). Patient wearing contact-lens–telescopic system.

while reading, as was demonstrated by his ability to read 48 words of ordinary newspaper print per minute.

Several improvements or alternatives in the telescope described may at once strike the reader. To produce a thinner lens the objective may be ground from high-index lead glass. However, such a glass would not weigh less, and would scratch more easily. A bifocal objective spectacle may be conceived but a +6 or 8 D. addition would hardly be practical. The need for exact adjustment of the pupillary distance of each segment would further increase the difficulty. It also occurred to us that instead of a flat surface, a concave surface might be ground on the corneal segment of the contact lens in order to yield a stronger eyepiece. This was discarded as impractical because accumulation of lacrimal secretion in the concave portion would probably destroy the optical effectiveness, and such a lens would be most difficult to grind.

The contact-lens–telescopic system is indicated in the same types of visual impairment as is the ordinary telescopic lenses. In those patients whose vision is permanently reduced from high myopia or a macular lesion they are most useful and satisfactory.

Summary

A method for constructing a telescopic spectacle by using a contact lens with a flat corneal segment as an eyepiece and a strong positive lens in an ordinary spectacle frame as the objective has been described. The patient for whom this contact-lens–telescopic system was prescribed was able to see as well at distance with it as with his Zeiss telescopic lens, and was able to read better. The cosmetic disfigurement was much less than with the Zeiss telescope and the field of vision was not restricted.

References

[1] Bruner, A. B. Telescopic lenses as an aid to poor vision. Amer. Jour. Ophth., 1930, v. 13, Aug., p. 667.
[2] Dallos, J. Contact glasses, the "invisible" spectacles. Arch. of Ophth., 1936, v. 15, April, p. 617.
[3] Boeder, P. Power and magnification-properties of contact lenses. Arch. of Ophth., 1938, v. 19, Jan., p. 54.
[4] Levy, A. H. Telescopic spectacles. Brit. Jour. Ophth., 1929, v. 13, Dec., p. 593.
[5] ——. Telescopic spectacles. Brit. Med. Jour., 1928, v. 2, Sept., p. 438.
[6] Berliner, M. A new type of telescopic lens. Arch. of Ophth., 1936, v. 16, Oct., p. 649.
[7] Eggers, H. Inexpensive telescopic spectacles. Arch. of Ophth., 1933, v. 10, p. 515.
[8] Hartinger, H. Special types of optically worked contact glass. Zeit. f. Ophth. Optik, 1932, v. 20, no. 5, p. 129.
[9] Gradle, H., and Stien, J. Telescopic spectacles and magnifiers as aids to poor vision. Trans. Sec. Ophth., Amer. Med. Assoc., 1924, p. 262.
[10] Obrig, T. E. Fitting of contact lenses for persons with ametropia. Arch. of Ophth., 1937, v. 17, June, p. 1809.
[11] Verhoeff, F., and Grossman, H. Pathogenesis of disciform degeneration of the macula. Arch. of Ophth., 1937, v. 18, Oct., p. 561.

VITAMIN THERAPY IN OPHTHALMIC PRACTICE

Joseph Laval, M.D.
New York

Much has been written about the importance of vitamins in general medicine, and three reviews have appeared in ophthalmic literature. It must of course be realized that much of what follows will be only suggestive and show certain trends and indications as to the uses of the various vitamins in ophthalmic practice. Definitely positive statements and findings will be infrequent, but some general conclusions will be self-evident.

Vitamin A

This vitamin is best associated in our minds with cod-liver oil and recently with haliver oil. It is present in good amounts in eggs, butter, milk, cream, and such dairy products. A deficiency of this vitamin manifests itself clinically first, as night blindness (hemeralopia) and later as xerophthalmia. Recently, dark-adaptation tests in early and beginning A avitaminosis have been advocated, but as yet they are not sufficiently standardized to warrant positive statements. It has been shown that the visual purple of the retina after exposure to strong sunlight regenerates slowly in the eyes of rats on a diet deficient in vitamin A (Fridericia and Holm) and it has also been proved that the normal retina is rich in vitamin A (Holm, also Yudkin, Kriss, and Smith). Vitamin A has been used to cure xerophthalmia and the hemeralopia due to depletion of vitamin A with, of course, brilliant results, as witness the cases of the fishermen of Labrador as reported by Aykroid, and the cases of the natives of the Philippine Islands reported by Gamboa. However, vitamin A has been used for the hemeralopia due to retinitis pigmentosa with no improvement at all, as was reported by me in 1933 (Levine).

Vitamin A is being used also in the conjunctival sac with massage into the cornea in cases of keratitis dystrophia, but the results are variable. Some authors report brilliant results, others no improvement. In the writer's own experience there were some cases in which it seemed to do

much good, others in which irritation and aggravation of the symptoms resulted.

In certain cases of blepharitis and conjunctivitis in children there is no doubt that malnutrition is a factor, and here large doses of cod-liver oil are beneficial. Pillat reports the changes in Chinese eyes as a result of vitamin-A deficiency and mentions night blindness, dryness of the conjunctiva, Bitot's spots, xerosis, and even keratomalacia, meibomitis, hordeolum, and blepharitis. Vitamin A is also of use in phlyctenular keratoconjunctivitis and other related conditions of scrofulous origin. In women a low-grade chronic conjunctivitis with mild photophobia may be due to A avitaminosis as a result of dieting in the effort to keep a streamlined figure. Langdon described the case of a young woman who used no butter, eggs, or milk and had a corneal dystrophy which cleared up on the administration of cod-liver oil.

When depletion of vitamin A is advanced, changes in the lacrimal gland result with diminution and even entire loss of lacrimation. Mori showed that these changes in the lacrimal gland were responsible for conjunctival changes in xerophthalmia, which then gave rise to the corneal changes. Yudkin, in similar experiments, drew different conclusions as to which lesions arose first, but Wolbach and Howe demonstrated that all the changes are due to a replacement of the columnar epithelium by stratified squamous epithelium going on to keratinization.

In diabetics there may be an increased demand for vitamin A, as certain experiments by Ralli *et al* showed. In depancreatized dogs (artificial diabetes) it was found that definite corneal epithelial changes occurred of the same type as in vitamin-A deficiency. These corneal changes were prevented by feeding cod-liver oil to the depancreatized dogs. Apparently, in these diabetic animals the demand for vitamin A is increased.

Vitamin B

This vitamin is found in such foods as meats, potatoes, and green vegetables, but is most concentrated in yeast. It has various complex components the most important of which are B_1, the antiberi-beri component (thyamine), and B_2 or G, the antipellagra portion (riboflavin), the absence of the latter causing cataracts in rats. However, for clinical purposes it is to be remembered that concentrated yeast tablets as marketed by any of the reliable pharmaceutical firms contain B_1 and B_2 in high amounts. (For a complete study of the vitamin-B complex see the article by E. M. Nelson in the Journal of the American Medical Association, 1938, v. 110, Feb. 26, no. 9, p. 645.)

In the Archives of Ophthalmology for December, 1934, the writer reported a case of bilateral acute optic neuritis with marked diminution of vision associated with pellagra. (It is known that the peripheral neuritis of pellagra and of beriberi is due to the lack of vitamin B_2 and B_1, respectively.) In this case the pellagra and optic neuritis both were cured, even though alcohol was taken in fairly large amounts daily, because yeast was administered in large doses, thus supplying the necessary quantity of vitamin B_2. In a case of toxic amblyopia due to alcohol, recently observed, vision was improved from hand movements to 20/20 for each eye in a period of two weeks by administering vitamin B_1 and B_2 in the form of yeast tablets, even though some alcohol was taken daily. Carroll reported the same results in a series of cases in 1937, and the present writer quoted Shastid's experience with cases of optic neuritis cured by vitamin B_1 and B_2. In all cases of retrobulbar neuritis, toxic amblyopia, and optic neuritis—whether due to nasal-sinus

infection, alcohol and tobacco, or multiple sclerosis—it would seem advisable to use concentrated yeast tablets to supply large amounts of vitamin B_1 and B_2.

Similarly, in cases of incipient cataract the same therapy should be carried out, for riboflavin (anticataractous for rats) is present in the concentrated yeast tablets. Furthermore, there is sulphur in these yeast tablets (HS-cystine), and some connection between the sulphur content of fish lenses and the prevention of human cataracts has been reported by Shropshire. A further connection between cystine and the formation of galactose cataracts in rats has also been shown; this will be discussed under vitamin C.

Vitamin C

This vitamin is found in the citrus fruits—orange, grapefruit, lemon, and lime. The pure crystal in the form of cevitamic acid has been prepared.

According to Bellows and Rosner, "In normal eyes the lens and aqueous are rich in vitamin C, in the cataractous lens the amount of vitamin C is diminished or entirely absent, and the aqueous of an aphakic eye contains only a slight amount of this substance. Experiments were performed which showed a definite reduction in the vitamin-C content of the blood in persons with cataract. Furthermore, vitamin C is absorbed from the conjunctival sac into the anterior chamber. Presumably this takes place through the cornea." These investigators also proved that the substance in the lens which reduces a special dye (sodium 2, 6, di-chlorobenzenone-indophenol) is entirely vitamin C, and not any other reducing substances such as sulphhydryls. Bellows went still further and showed that the cataract produced in the albino rat by lactose (Mitchell and Dodge, Mitchell and Cook) and by galactose (Yudkin and Arnold) is due to a loss of the sulphhydryl content of the crystalline lens which can be delayed by cystine and to a lesser degree by vitamin C in this type of cataract. Accordingly, it would seem that in cases of incipient cataract vitamin B_1 and vitamin B_2, to supply the cystine, and also vitamin C should be administered. In the writer's own practice this procedure has been carried out for the past four years, but of course no conclusions should be drawn as yet.

Bietti believes that the decrease in the amount of vitamin C in the aqueous and in the cataractous lens is the result of the lens changes and not the cause. He was unable to prevent or influence naphthaline cataract by administering vitamin C.

In 1932 Szent-Györgyi and Svirbely in Hungary proved that hexuronic acid is vitamin C. At the same time King and Waugh of the University of Pittsburgh showed that a crystalline compound which they isolated from lemon juice was identical with hexuronic acid. Szent-Györgyi found that the best source of vitamin C was the peppers grown in Hungary. However, patients with purpura were relieved by the juice of peppers or of citrus fruits but not by the pure crystalline vitamin C. On further investigation it was found that citrus fruits and the peppers contain a second substance controlling the permeability of capillaries and this substance is called vitamin P. The symptoms of scurvy seem to be due to a lack of both vitamin C and P.

Accordingly, it would seem advisable for ophthalmologists to utilize the juice of citrus fruits in large doses in attempting to control intraocular hemorrhage whether due to trauma (operative or other) or to some metabolic disease such as diabetes. Yudkin recently advocated the same therapy and seemed quite encouraged by its results. The present writer has also tried it in several cases of intraocular hemorrhage, both postoperative

and metabolic, and feels encouraged enough to continue with its use.

Vitamin D

The use of vitamin D in ophthalmology has been limited to cases of myopia in the hope that this vitamin plus calcium would cause an increase in the amount of calcium in the sclera and thus prevent further stretching. Recently the writer reviewed this subject and gave the results of clinical experiments which showed no favorable influence on myopia after vitamin-D therapy. However, it should be borne in mind that the prescribed dosage of 10 minims daily may have been insufficient. Inasmuch, however, as this is the dosage for cases of rickets we may assume that it is sufficient for cases of myopia investigated clinically. The use of vitamin D in keratoconus has also been suggested.

Summary

It is suggested that in cases of the following vitamin deficiencies it might be theoretically valuable to prescribe the following treatment:

1. Vitamin A—Prescribe a tablespoonful twice daily in cases of poor dark adaptation, phlyctenular kerato-conjunctivitis, photophobia, and low-grade conjunctivitis in women who are on a slenderizing diet or in cases of other corneal and conjunctival lesions in which the history shows a lack of vitamin-A intake.

2. Vitamin B (B_1 and B_2)—Prescribe eight yeast tablets daily of the brewers'-yeast type put up by any of the reliable pharmaceutical firms, or the powdered form may be ordered. This is to be used in cases of incipient cataract, in optic neuritis, retrobulbar neuritis, and also in toxic amblyopia.

3. Vitamin C—Order the juice of at least two large oranges or one grapefruit daily in cases of incipient cataract. (This is plus the brewers' yeast which is also to be taken daily.) In cases of intraocular hemorrhage order the juice of four lemons daily.

136 E. Sixty-fourth Street.

Bibliography

Adler, F. H. Ocular disorders in deficiency diseases. Arch. of Ophth., 1927, v. 56, Nov., p. 593.

Aykroyd, W. R. Beri-beri and other food deficiency diseases in Newfoundland and Labrador. Jour. Hyg., 1930, v. 30, Aug., p. 357.

Bellows, J. Biochemistry of the lens V. Cevitamic acid content of the blood and urine of subjects with senile cataract. Arch. of Ophth., 1936, v. 15, Jan., p. 78.

Bellows, J., and Rosner, L. Biochemistry of the lens VIII. A new proof of the presence of vitamin C in the crystalline lens. Arch. of Ophth., 1936, v. 16, Aug., p. 248.

Bietti, G. Vitamin C in the ocular tissues and liquids: Its relation to the biology of the lens. Boll. d'Ocul., 1935, v. 14, Jan., p. 3.

Carroll, F. D. Importance of diet in the etiology and treatment of tobacco alcohol amblyopia. Arch. of Ophth., 1937, v. 18, Dec., p. 948.

Day, P. L., Langston, W. C., and O'Brien, C. S. Cataract and other ocular changes in vitamin G (B_2) deficiency. Amer. Jour. Ophth., 1931, v. 14, Oct., p. 1005.

Dodge, W. M., Jr. Histopathologic characteristics of nutritional cataracts in the white rat. Arch. of Ophth., 1935, v. 14, Dec., p. 922.

Fridericia, L. S., and Holm, E. Experimental contributions to the study of the relation between night blindness and malnutrition. Amer. Jour. Physiol., 1925, v. 73, p. 63.

Gamboa, L. Deficiency in vitamin A and nyctalopia. Jour. Philippine Islands Med. Assoc., 1930, v. 10, June, p. 235.

Holm, E. Demonstration of vitamin A in retinal tissue. Acta Ophth., 1929, v. 7, p. 146.

Kendall, A. I., and Gifford, S. R. Trachoma and avitaminosis. Arch. of Ophth., 1930, v. 4, Sept., p. 322.

King and Waugh. In E. C. Kendall's "The work of Professor Szent-Györgyi, recipent of the Nobel Prize in Physiology and Medicine," The Scientific Monthly, 1938, Jan., pp. 99-100.

Lambert, R. A., and Yudkin, A. M. Changes in the para-ocular glands accompanying the ocular lesions which result from a deficiency of vitamin A. Jour. Exper. Med., 1923, v. 38, July, p. 25.

Lane, L. A. Practical points in ophthalmic practice—A study of recent food researches. Jour. Amer. Med. Assoc., 1932, v. 98, Feb. 27, p. 726.

Langdon, H. M. A case of dystrophy of the cornea which disappeared on the administration of fats. Amer. Jour. Ophth., 1928, v. 11, May, p. 386.

Langston, W., Day, P. L., Cosgrove, K. W. Cataract in the albino mouse resulting from a deficiency of vitamin G (B_2). Arch. of Ophth., 1933, v. 10, Oct., p. 508.

Laval, J. Myopia and vitamin D. Arch. of Ophth., 1938, v. 19, Jan., p. 47.

Levine, J. Is retinitis pigmentosa due to vitamin deficiency? Arch. of Ophth., 1933, v. 9, March, p. 453.

———. Pellagra as a cause of optic neuritis. Arch. of Ophth., 1934, v. 12, Dec., p. 902.

Mitchell, H. S. Proc. Soc. Exper. Biol. and Med., 1935, v. 32, p. 971.

Mitchell, H. S., and Cook, G. M. Galactose cataract in rats. Arch. of Ophth., 1938, v. 19, Jan., p. 22.

Mitchell, H. S., and Dodge, W. M. Jour. Nutr., 1935, v. 9, p. 37.

Mori, S. The changes in the para-ocular glands which followed the administration of diets low in fat soluble vitamin A. Bull. Johns Hopkins Hosp., 1922, v. 33, Oct., p. 357.

Nelson, E. M. The chemistry of the vitamin B complex. Jour. Amer. Med. Assoc., 1938, v. 110, Feb. 26, p. 645.

O'Brien, C. S. Experimental cataracts in vitamin G deficiency. Arch. of Ophth., 1932, v. 8, Dec., p. 508.

Pillat, A. The main symptoms of the eye in vitamin A deficiency in adults. Nat. Med. Jour. China, 1929, v. 15, pp. 585 and 614.

Ralli, E. P., Gresser, E. B., and Flaum, G. Effect of experimental diabetes on the cornea of dogs: Its relationship to the administration of vitamin A. Arch. of Ophth., 1935, v. 14, Aug., p. 253.

Shastid T. H. Optic neuritis and vitamin B. Amer. Jour. Ophth., 1929, v. 12, Nov., p. 903.

Shropshire, R. F. Fish lens protein and cataract. Arch. of Ophth., 1937, v. 17, March, p. 505.

Szent-Györgyi and Svirbely. In E. C. Kendall's "The work of Professor Szent-Györgyi, recipient of the Nobel Prize in Physiology and Medicine," The Scientific Monthly, 1938, Jan., pp. 99-100.

Tassman, I. S. Dietary deficiency and ocular disease. Arch. of Ophth., 1932, v. 8, Oct., p. 580.

———. The cataractous lens: Experimental and clinical studies. Arch. of Ophth., 1938, v. 19, Jan., p. 114.

Williams, R. R. The chemistry of thyamin (vitamin B_1) Jour. Amer. Med. Assoc., 1938, v. 110, March 5, p. 727.

Wolbach, S. B., and Howe, P. R. Tissue changes following deprivation of fat soluble vitamin A. Jour. Exper. Med., 1925, v. 42, Dec., p. 753.

Yudkin, A. M. Experimental xerophthalmia in rats. Proc. Soc. Exper. Biol. and Med., 1922, v. 19, p. 375.

———. The presence of vitamin A in the retina. Arch. of Ophth., 1931, v. 6, Oct., p. 754.

———. Ocular manifestations of the rat which result from deficiency of vitamin A in the diet. Jour. Amer. Med., Assoc., 1922, v. 79, Dec. 30, p. 2206.

———. Vitamins in treatment and prevention of ocular diseases. Arch. of Ophth., 1938, v. 19, March, p. 366.

———. Clinical evidence of necessity of vitamins for function of ocular tissue. Arch. of Ophth., 1935, v. 14, July, p. 112.

Yudkin, A. M., and Arnold, C. H. Cataracts produced in albino rats on a ration containing a high proportion of lactose or galactose. Arch. of Ophth., 1935, v. 14, Dec., p. 960.

Yudkin, A. M., Kriss, M., and Smith, A. H. Vitamin A potency of retinal tissue. Amer. Jour. of Physiol., 1931, v. 97, July, p. 611.

LECTURES ON MOTOR ANOMALIES*

V. Development and course of strabismus

A. Bielschowsky, M.D.
Hanover, New Hampshire

The answer is, that the result of the therapeutic procedures depends to a high degree upon the kind of retinal correspondence that is present in the individual case. The prospect of restoring binocular single vision by correcting the faulty position of the eyes will, of course, be much better if the normal correspondence has been proved to be intact. Further, most ophthalmologists will recall patients with alternating squint who had never noticed diplopia but suffered tremendously from it after the squint had been corrected or reduced by operation. The diplopia in some of these cases is a paradoxical one; that is, the distance between the double images points to the difference between the present and the former position of the eyes. Thus, for instance, a patient whose convergent squint of 25 degrees had been reduced to a convergence of 5 degrees will see crossed double images of about 20 degrees; or, if a divergent squint of 30 degrees had been reduced to 5 degrees, will see homonymous double images of about 25 degrees. Sometimes the diplopia disappears after a few days due to a reëstablishment of suppression. In other cases the diplopia can be very persistent and cause a good deal of trouble to the patient as well as to his doctor, who might be reproached by the patient for not having told him before the operation about a possible diplopia afterwards. One can prevent such an unpleasant situation by ascertaining whether or not anomalous retinal correspondence has developed during the period of squinting. In repeated examinations (which must be made in any case where an operative procedure seems to be necessary), many patients, if tested with afterimages, will find to their surprise that they no longer see the afterimages separated from one another but as forming a cross. This will indicate that the anatomic substratum of normal retinal correspondence, although present, is not functioning under the ordinary conditions of seeing, in which the substitute is more convenient. But in a dark room, where the patient sees nothing but the afterimages, the dormant normal correspondence has a better chance to arouse and maintain itself against or beside the anomalous correspondence. Of course, in using the afterimage test one must avoid any influencing suggestion. The patient must be told, before the beginning of the test, only that he will see a vertical and a horizonal afterimage of the glowing filament and that he must carefully watch the position of both lines relative to each other so that he may be able to reproduce it by means of a simple drawing. If these conditions are fulfilled the result of the test will be unequivocal.

In cases of alternating squint one should not fail to ascertain how the eyes react to simultaneous stimulation of both the foveae by means of the phorometer or, better still, of the haploscope. If simple drawings, such as a circle and a square, respectively, are presented to either eye separately, the drawings will be fused or will cover each other, provided the correspondence functions normally. But in some cases it is very difficult or even impossible to make the patients see both images situated on the

* From The Dartmouth Eye Institute, Dartmouth Medical School. Read before the Seventh Annual Mid-Winter Clinical Course of the Research Study Club, Los Angeles, California, January, 1938.

maculae at the same time. They will see only one of the images either with the right or the left eye, the other image being suppressed (alternating uniocular vision). By shifting the image in the squinting eye, particularly in the vertical direction, it can more readily be brought to the patient's consciousness and it may remain visible for a while if it is now slowly brought toward the macula. But it will disappear before it has reached the latter and, if the shifting is continued very slowly, it will suddenly appear beyond the second image, or below or above it. Occasionally the images may even be made to touch or partly overlap, but one will not attain fusion or a perfect covering of the two images. This peculiar behavior can be explained only by assuming that the patient changes the angle of squint, instinctively trying to find a position in which the images would not be situated on corresponding retinal points, because two identical macular images localized in the same direction without being fused may, in the case under discussion, cause such an uncomfortable feeling that the patient, without knowing it, endeavors to avoid it by changing the angle of squint. To ascertain this behavior before any operation is performed is very important. For, in such cases there will very probably be a disturbing diplopia after the squint is removed or reduced to a minimum. Usually it disappears sooner or later. But one cannot tell how long the diplopia will last in the individual case. Therefore one ought never to omit discussing this possibility with every patient having alternating squint who shows symptoms of anomalous correspondence, and particularly the unsteadiness of the angle of squint just described, which would suggest the phenomenon of horror fusionis. The appearance of this phenomenon depends not unconditionally on the existence of an anomalous correspondence. I have also seen it in cases in which the double images corresponded perfectly to the small angle of squint that had remained after one or several operations. Horror fusionis may be due to a defective development of the sensorial apparatus in the occipital lobes. In such cases there is neither a normal nor an anomalous but no sensorial correspondence at all because the two eyes of the patient do not present the halves of a single organ, Hering's "Doppelauge," but two organs which, in regard to the sensorial apparatus, are independent of each other. But such cases, I am sure, are extremely rare. Horror fusionis may be produced by a protracted disuse of one eye, or arise due to a severe nervous shock, and the fusion faculty may be capable of being developed by proper training and a most careful removal of anything that may cause a difference between the visual function of both eyes.

Passing on to the development and the course of the different kinds of strabismus, I am going to discuss, first, the most frequent and, with regard to the therapeutics, the most important form; that is, convergent squint. It arises, as a rule, in early childhood. Only in a small percentage of the cases does it begin after the sixth year, following a disease or injury reducing the vision of one eye; sometimes it is due to a protracted occlusion of one eye, if it had been bandaged for a couple of days. I have seen some cases of this kind, one of them in a boy of 14 years who came to our clinic some time ago because of a chalazion in the right upper lid. After the extirpation the right eye was bandaged for two days. When the eye was uncovered he complained of diplopia. There was a manifest convergent squint of 18 degrees. The double images were uncrossed and their separation subtended exactly the objective angle of squint. He had a hyperopia of 5 diopters, which had never been corrected, since

the boy had full vision of both eyes and had never noticed any trouble previously. His brother had been operated on for permanent convergent squint two years before. Even after atropine had been given and the refractive error had been corrected the deviation did not disappear. But when the retinal images were shifted from the disparate to corresponding points by means of prisms or the haploscope there was fusion with depth perception; in addition, an unusually high amount of aniseikonia was determined by Professor Ames. The images of the two eyes could be equalized by meridional size lenses of 8 percent axis 90 degrees before the right and 6 percent axis 180 degrees before the left eye. The patient had binocular single vision when he wore before each eye a combination of + 4.00 and 10$^{\Delta}$, base out, with an aniseikonic correction in a temporary frame. After three days he reported that he had broken the glasses which were in the left side of the trial frame, but nevertheless he had binocular single vision when wearing just one set of glasses before the right eye. I found the convergent squint reduced from 18 degrees to 5 degrees. The aniseikonia was likewise reduced considerably. There was still spontaneous diplopia but, in order to get binocular single vision he needed only 2$^{\Delta}$, base out, before each eye in addition to the hyperopic correction. After three days he discarded the glasses, since he had found that he had binocular single vision without them. When examined he showed an esophoria which, after suspension of fusion, became manifest and gradually increased up to 9 degrees with and without the glasses correcting the hyperopia. Vision was 20/15 with and without the glasses.

The case is a very instructive illustration of the different factors which have been discussed as being responsible for the development of squint and particularly of the decisive role played by the fusion apparatus in the etiology of squint. The combination of three important etiological factors—namely, inherited disposition, excessive accommodation induced by hyperopia of more than 5 diopters, and a considerable esophoria based on an anomalous position of rest—brought about neither a permanent nor even a periodic convergent squint as long as it was counteracted by the fusion apparatus in the interest of binocular single vision. But why did the fusion apparatus fail to overcome the tendency to squint for a considerable length of time after the bandage had been removed from the right eye? Since the correction of the hyperopia did not reduce the angle of squint more than a few degrees, it is evident that in this case the excess of accommodation was an etiologic factor of less importance than the convergent position of rest which, according to the results of later examinations, amounted to 9 degrees; that is, half the angle of the convergent squint which was found after the removal of the bandage. The other half is to be attributed to an excessive convergence innervation due, very likely, to so-called diplopiaphobia. It is not unusual that a person, noticing double images for the first time and trying to fuse them without success, will find instinctively that the double images are less disturbing if their separation is increased. The only voluntary innervation which may serve that purpose—by altering the position of the eyes relative to each other—is the convergence innervation. Whether or not diplopiaphobia plays such an important role in the etiology of squint as some authors (Duane, van der Hoeve) believed, is still an open question. In my opinion a convergent squint is to be attributed to diplopiaphobia only in a comparatively small number of cases. Our patient's convergence spasm subsided gradually when about half of the deviation was compensated for by means of prisms. The re-

maining half was overcome by the patient's powerful fusion apparatus. Without going into details of this most interesting case, I just want to mention the fact that the high amount of aniseikonia that was found during the period of convergent squint decreased almost exactly in proportion to the decrease of the deviation. At present, as soon as the latter is again evoked and brought up to the previous amount by an adequate haploscopic arrangement, a corresponding increase of the meridional aniseikonia is reëstablished.

The results to be expected from a rational treatment in cases in which convergent squint has arisen relatively late, are much better than in the other group because of the existence of a fully developed normal retinal correspondence. Of course, the attainment of the ideal result, the restoration of binocular single vision depends also in these cases on the vision of the squinting eye. If an existing amblyopia can be improved, fusion exercises by means of the phorometer or the haploscope or other orthoptic apparatus will, in such cases, have the very best chances, sometimes even without any operation; for instance, in cases in which an original esophoria had been transformed into a manifest convergent squint in consequence of an occlusion of one eye. In the main group, patients with a convergent squint existing since early childhood, the ideal result—namely, binocular single vision—can be obtained in only a much smaller percentage of the cases. That is due partly to the frequent amblyopia of the squinting eye, partly to a persistent anomalous correspondence or a deficiency of the fusion apparatus, either congenital or acquired in earliest childhood, so that very frequently one must be satisfied with the removal of the cosmetic disfigurement. The earlier the squint can be treated the better the prognosis. By bandaging the fixating eye for weeks or months a considerable improvement of the vision of the squinting eye may be achieved if the amblyopia is not of an organic but of a functional origin. The second task in such a case—a careful correction of any refractive error is presupposed—would be to overcome the supression tendency of the squinting eye. To accomplish this the child must be at least three years old or more, so that it can tell about the double images and the images seen in the stereoscope or amblyoscope. It is not very difficult to find out whether the position of the double images relative to each other is due to an anomalous or to the normal retinal correspondence. In the latter case one may try to obtain fusion by means of prisms; Sattler reported very gratifying results in this way even when he had to prescribe prisms up to 20$^{\Delta}$ for either eye. Their strength can be reduced after some weeks or months if fusion is obtained. If not, suppression and amblyopia of the squinting eye will return unless the occlusion of the dominant eye is continued.

In cases of a constant deviation of more than 20 (arc) degrees, when a complete relaxation of accommodative effort, first by means of atropine, then by a full correction of hyperopia, has failed to bring about any improvement in the condition or has removed only a small fraction of the deviation, an operation must be considered. As to the earliest age at which a child with a convergent squint can be operated on, definite rules cannot be given. The patient should not be operated on before glasses, possibly combined with prisms, have been worn for several months and the angle of squint has been found to be rather constant during repeated examinations. In quite a number of cases the squint decreases gradually and, while the children are growing, finally disappears, mainly because of the change of the topographic anatomic conditions which favor the tendency of the

eyes to diverge. As long as a decrease of the angle, even though it be small, can be ascertained, one should wait. But if an angle of more than 10 (arc) degrees is the same after six months of observation, or has possibly increased, I do not hesitate to operate on a child of about four years of age. The older the patients, the sooner an operation may be performed after the angle of squint has been found to be fairly constant in repeated examinations and after the nonoperative procedures have been tried without success. The prospects for these procedures will be better after the angle of squint has been reduced to a small fraction, provided that the amblyopia or a defective condition of the fusion apparatus does not present insuperable obstacles. There are cases in which binocular single vision and depth perception together with a certain amplitude of fusion sets in immediately and spontaneously after the operation. But in the majority of cases in which any good result is to be obtained at all, it takes some time until binocular single vision can be demonstrated after the angle of squint has been reduced to a residue of a few degrees by operation. Fusion training might help a good deal, but in the end it will depend on the condition of the cortical centers governing the fusion apparatus whether the functions of *perfect* binocular single vision, including depth perception and fusional amplitude, will be obtained or just fusion without depth perception or fusion only for near and not for distant objects because of a deficient fusional amplitude. These patients, as a rule, do not notice diplopia, availing themselves of the faculty acquired during the previous squint to suppress the images of one eye. The functional deficiency of the fusion apparatus can be improved by exercises only if it does not depend on a defective condition of its cortical centers. But even cases in which all the symptoms point to a congenital deficiency of the fusion apparatus may be cured by proper treatment, thus proving that they are functional anomalies, as in the following two examples.

A student, 20 years old, unusually ambitious and industrious, had been operated on three times by one of the most prominent ophthalmologists on account of a convergent strabismus that had arisen in earliest childhood. Seven weeks after the last tenotomy, diplopia developed to the great annoyance of the patient. He was a big, strong fellow, and apart from a very pronounced neuropathic habitus, perfectly healthy. Both refraction and vision were normal. There was a permanent convergent strabismus of 2 degrees for distance, but the double images were alternately homonymous and crossed, fluctuating greatly, up to an amount of 10 degrees. Apart from the convergent deviation the right eye was turned upward, the amplitude also fluctuating between 2 and 14 degrees, but the objectively measured vertical deviation always corresponding to the vertical separation of the double images. There was no sign of a paretic anomaly, the angle of squint being independent of the direction of view. All attempts with prisms and haploscope to get the double images fused failed, due to the continual changes of the angle of squint. Because of these conditions and in view of the very small deviation I doubted whether I should be able to relieve the patient of his diplopia; but since he insisted on my making a last attempt to remove his troubles I advanced his left external rectus, producing as an immediate result a divergence of 10 degrees. It decreased gradually and was finally transformed into a convergence of 2 degrees; that is, the same amount as before the operation. But the vertical divergence had disappeared and did not return. The horror fusionis remained, the patient saw now crossed, now uncrossed double images that could not be fused and were

annoying to such a degree that the patient after a month earnestly asked to have his original squint restored in order to be able to continue his studies. But I finally succeeded in persuading him to wait six weeks longer and to submit to regular stereoscopic exercises. Gradually, the fusion faculty was developed so that after six weeks the patient noticed double images only of small objects contrasting with the background, and at last acquired even a certain amount of depth perception. The ophthalmologist who supervised the exercises told me that during the exercises the patient always had to overcome enormous psychic inhibitions. He gave the impression as though he were actually looking for double images, but fortunately no longer found them.

This case is interesting in some respects. It confirms the experience gained in other cases of vertical divergence which, if they are due to intermittent nervous excitations should not be operated on, because they may disappear without any particular treatment if restoration of the fusion faculty can be achieved. It confirms the rule, the importance of which I have emphasized time and again that, in the face of apparent failures of squint operations, to the performance of which no objection could be raised, one must not be induced, in such cases, to precipitate procedures which would spoil the effect of the operation. One has to remind the patient that he must endure the troublesome condition for at least two or three months and that he must await the results of exercises. Finally, the case demonstrates that horror fusionis may not be caused by a congenital defect but may be due to a neurotic disturbance and can be cured even if it has existed for years and in spite of the various kinds of treatment that have been tried unsuccessfully.

Another extremely interesting case of horror fusionis was demonstrated by me at the meeting of the New England Ophthalmological Society in January, 1935, and was published in my article on "Congenital and acquired deficiencies of fusion" (American Journal of Ophthalmology, 1935, volume 18, October). The case has, in my opinion, a fundamental significance. The patient, who had been operated on several times on the lateral as well as the vertical ocular muscles because of a complicated squint, showed a slight deviation composed of a lateral, a vertical, and a torsional component, changing with the slightest alteration of view because of a paretic weakness of almost all the muscles of the right eye. There was, according to the direction of gaze and the position of the head, homonymous or crossed diplopia with positive or negative vertical divergence and conclination or disclination of the vertical meridians. Even for one and the same direction of gaze the deviation was very unsteady, due to spasms of the convergence and the vertical divergence innervations. Fusion could not be obtained by any method. Only after an unusually high amount of aniseikonia had been determined, and corrected by size lenses, could binocular vision with depth perception and a certain fusional amplitude be established. Aniseikonia has to be looked for in every case of weak or defective fusion because it may be an important etiologic factor, not only in so far as it involves an incongruence of the ocular images (such as ametropia or other conditions impairing the visual acuity), but because it may produce anomalous and nervous irritations as well as a weakening of the physiologic fusion mechanism, either in its sensorial portion by favoring suppression, or in the motor portion by checking the fusion innervation, on account of a mental antipathy toward the fusion of heterogeneous ocular images.

PARALLEL STUDY OF THE PATHOGENESIS OF RHINOGENOUS OPTIC NEURITIS AND OF SEROUS IRITIS

BÉLA WALDMANN, M.D.
Oradea (late Nagyvárad) Roumania

In the last score of years, the American medical literature has subjected the question of retrobulbar neuritis to a thorough study, but it is evident that opinions as to the origin of this disease are still divergent.

In 1906 I was so fortunate as to be present at the meeting of the Society of Hungarian Oculists, before which Onodi demonstrated for the first time his world-renowned preparations. He made a very deep impression on his audience and aroused their profound interest in this problem. His address was of great interest to me, and under its influence, during the course of the last three decades, I have paid especial attention to all those cases that could be brought into causal connection with the accessory nasal sinuses. Thus I have acquired a considerable amount of data which have now encouraged me to essay the solution of the pending questions from my own experience.

The relation of single cases of iritis to the mucous membrane of the nose was first mentioned by Ziem in 1887; namely, he observed in three instances that the iritis underwent a considerable improvement upon treatment applied to the simultaneously existing rhinitis. Kuhnt denied the possibility of the causal connection, and attributed the improvement to the fact that the cauterization of the mucous membrane of the nose, in a collateral way, diminished the hyperemia of the uvea, and thus brought about the improvement. The conflicting opinion of this prominent ophthalmologist buried the question until the present day, although, as will be apparent from the following comments, it well deserves a careful study.

A. OPTIC NEURITIS

Before entering into the discussion of the pathogenesis, I shall describe some types out of my own collection of cases, selecting those that are apt to support my conception.

Type 1. M. L., a female, aged 46 years, consulted me on March 29, 1928, complaining that six days ago she had gone to bed with perfect sight and next morning, upon awakening, noticed that she saw nothing with her right eye.

Examination: No objective vision in the right eye; light perception good. Vision in the left eye 5/5. Both eyegrounds were wholly normal. Suspecting that this might be a case of hysteria, I made control examinations which, however, yielded a negative result. The nervous system was unimpaired; a blood test, negative.

Diagnosis: Neuritis retrobulbaris.

Rhinoscopy: No secretion could be seen on the right side on account of hypertrophy of the turbinate. X-ray report was negative. Upon my strong recommendation next day the middle nasal turbinate was resected and the ethmoidal cells opened, whereupon abundant, purulent secretion was evacuated from all their sections and even from the sphenoidal recess. Subsequent to the operation vision rapidly improved and, after the lapse of two weeks, became completely normal.

Comment: In the present case the X-ray report was of no avail, and although vision had been rapidly reduced to the minimum, the eyegrounds displayed nothing abnormal. This form presents the most favorable prognosis, visual recovery taking place in a fairly short time.

Type 2. S. E., a female, aged 36 years, called on me on January 4, 1928, with the complaint that her sight in the left eye had abated during the last few days.

Examination: Vision in the right eye 5/5, in the left 5/30; in the left fundus the outlines of the papilla were indistinct, the blood vessels tortuous.

Diagnosis: Intraocular optic neuritis; that is to say, a mild papilledema. The nervous system was unimpaired; blood test, rhinoscopy, and X-ray studies were negative. Notwithstanding, I daily placed cocaine-adrenalin swabs into the left nasal meatus. Although this procedure resulted in the continuous evacuation of a pro-

fuse, muco-purulent secretion, vision nevertheless gradually became so reduced that on the 28th of January the patient could only count fingers from a distance of half a meter. Then I urged the opening of the ethmoidal cells, and it was found that the mucous membrane of the left ethmoidal sinus showed polypoid degeneration. From the day of the operation the sight gradually improved and on the 18th of April it had become 5/7. Four years subsequently the same process was repeated in the other eye, following a similar course.

Comment: From this case, which represents the moderately severe form, the lesson can be drawn that the failure to obtain results by applying swabs by no means disproves the rhinogenous origin of the neuritis, but, on the contrary, in such instances an operation is urgently indicated.

Type 3. M. I., a female, 21 years of age, consulted me on December 22, 1937, complaining about considerably impaired vision in her left eye.

Examination: Right eye unimpaired; in the left fundus the papilla was greatly elevated (3 D.), its outline wholly blurred, but the filling of the blood vessels scarcely deviated from the normal.

Diagnosis: Papilledema in a high degree. Vision from a distance of 2 meters is ability to count fingers. The blood test was negative; rhinoscopy revealed the presence of abundant purulent discharge in the middle meatus. I suggested to the patient to have her nose treated, but owing to the approaching Christmas holiday she refused to do this and went home. In five weeks' time she again appeared, her vision having diminished to counting fingers at half a meter, and the optic nerve was by way of becoming atrophied. She refused to undergo treatment at this time also.

Comment: This case represents the most serious form of optic neuritis; namely, one in which the elevation of the disc is pronounced. The fibers of the optic nerve are very sensitive to pressure and quickly atrophy.

PATHOGENESIS

It is unnecessary to mention in detail the pertinent American literature, and I shall therefore confine myself to enumerating only those authors whose writings have been accessible to me; namely, W. L. Benedict, E. Campbell, C. Crane, S. Gifford, L. White, and the medical staff of the Johns Hopkins Hospital under the direction of A. Woods. Although these writers have brought the very best of their knowledge to the clearing up of this question, yet according to the results of H. Campbell the opinions are so contradictory that they may be conducive to considerable confusion in the reader. Some authors regard the disease of the optic nerve to be a septic focus originating in the teeth or the tonsils. A considerable number of ophthalmologists hold that the provocator is the collateral edema, and there are only a few who take for granted that the source of the affection is in the pressure conveyed through the thinned bony separating wall. The assumption that the inflammation in the canal is brought about by the direct spreading of the infection from the sinus has most adherents. This thought gained strong support in the discovery of Herzog, according to which the medullary cavities in the bony wall of the accessory sinuses lead directly towards the mucosa, likewise towards the dura mater, and thus provide a direct path for the passage of the causative agents. Hajek also concurred in this concept of Herzog, but in my opinion this cannot be applied in cases of optic neuritis, but only in those of serous iritis, and for the following reasons:

First: If pathogenic agents were to penetrate into the optic canal, their propagation would not be restricted to the canal, but they would migrate farther either into the brain or into the eye, where they would provoke an inflammation such as we generally see at postmortem examinations of those cases of serous iritis and sympathetic ophthalmia in which the uveitis of the fellow eye was preceded by an optic neuritis, as was shown by the animal experiments of the younger Velhagen. To my knowledge no mention has been made in the literature of such an instance of optic neuritis of rhinogenic origin followed by uveitis.

Second: In optic neuritis the pathogenic agent could not even migrate from the diseased sinus, for in my cases, as also in those under the observation of Campbell and Gifford, rhinoscopy often established a diagnosis of chronic hypertrophic inflammation, or empyema. In these cases the strepto- and staphylococcus, and, according to the finding of James J. Moore, even the Bacillus pyocyaneus may be encountered in the accessory cavities. Against this peril the organism raises a defense, preventing the escape of these dangerous pathogenic agents into the canal by forming a defensive wall; for otherwise most individuals suffering from empyema would die of meningitis. According to Hajek the edema in such cases is restricted only to the upper strata of the mucosa, while the rest down to the periosteum become transformed into a coarse, fibrous connective tissue. White histologically examined nine cases and similarly found a fibrous thickening of the mucosa which sometimes involved even the bone. According to my own observations, this is a dam that is impervious to the pathogenic agents; therefore, they cannot play a role in bringing about neuritis, and the whole process may be regarded as aseptic.

Thus, in bringing about optic neuritis the following two factors may play a part: either direct pressure, or collateral edema.

In a closed empyema it is easily possible, that in connection with the disintegration of the mucosa and its purulent necrosis, gases of decomposition develop that may be conducive to the tightening of the bony wall. If the latter is thinner than normal and at the same time the abnormal course of the canal brings it nearer to the sinus, the rise of pressure in the cavity may be directly conveyed through the bony wall to the soft tissues in the canal. Thus these may suffer compression and give rise to the various grades of neuritis: (a) in mild compression, retrobulbar neuritis (normal fundus); (b) in moderate compression, intraocular neuritis; (c) in compression of a higher degree, papilledema.

The clinical symptoms of the cases I have described indicate the existence of these three grades.

Besides the enumerated American authors, the role of the collateral edema has been corroborated also by Traquair, Letchworth, and recently by Sakaye Kitahara, who says that in the neighboring formations this is produced by the purulent imbibitions of the sinus. To this edema can be traced those cases in which the deterioration of vision sets in suddenly; similarly rapid, also, is its return, as was noted in my case, type 1. An edema may appear in such a short time, but not the disturbance caused by the intrenched pathogenic agents.

By the aid of roentgenograms, L. White established the existence of canals of different gauges; in general, the calibers were of 4 to 6 mm., and from this he drew the conclusion that canals of narrower caliber are more apt to promote the occurrence of neuritis than those that are wider. Apart from the fact that the roentgenograms of the accessory cavities, in general, are disappointing, I consider White's conclusions to be erroneous, for the fluid in the intervaginal cavity compensates for the 1 to 2-mm. difference, the space of the canal being filled in every instance. Thus the pressure exerted upon its wall, or the edema occurring in it, in the same way creates a narrowness of space, whether its caliber is 4 or 6 mm. Besides, Endre Luzsa* on the basis of numerous screening examinations states that roentgenograms taken of one and

---

* State Eye Hospital, Budapest; Director, Professor Imre.

the same canal at different planes may display various dimensions, because an infinitesimal change in the position of the tube, of the film, and of the skull is apt to influence the outlines of the shadow, and therefore we cannot obtain absolute values. For this reason I shall not follow White's advice—namely, that nasal treatment is superfluous in the case of canals of 5 to 6-mm. diameters—because according to him, the optic nerve is not endangered, and spontaneous recovery can be awaited without risk.

From the foregoing it can easily be explained why rhinogenous optic neuritis is so rare, although the inflammation of the accessory cavities is of frequent occurrence. This is emphasized by several authors as a counter-argument to the theory of those who believe in the rhinogenous origin of single neuritises; namely, that for the development of an optic neuritis, two conditions are necessary: first, the abnormal thinness of the bony separating wall; second, an anomaly in the course of the optic canal which brings about a closer relationship between the optic nerve and the adjacent sinus,** and thus direct pressure, or the evolution of a collateral edema becomes possible. As the simultaneous presence of these two anomalies may be regarded as rare, accordingly also optic neuritises connected with them are strikingly rare, but this does not justify a doubt as to the existence of this pathologic form. Thomson also acknowledges optic neuritis to be a relatively rare disease, but he accentuates its great importance, requiring quick and energetic treatment.

Thus it is not justifiable to omit or postpone the treatment of the accessory cavities because in some cases the condition healed spontaneously. According to Hajek, a partial or entire impediment to the evacuation of the secretion forms one important cause of the chronicity of the sinus affection. In this the composition of the secretion is significant, for according to him a thick discharge may be impeded even if the efferent duct is fairly wide. It is no wonder, therefore, that in optic neuritis, in the presence of a closed empyema, radical operation in the nose must often be performed because the swab had proved unsatisfactory. This was realized in my example of type 2, which, on the other hand, was under my constant control during the latter 10 years and in spite of the fact that optic neuritis occurred on both sides, no symptoms of multiple sclerosis were present. On the other hand, Meller believes that if, subsequent to an optic neuritis, a multiple sclerosis should make its appearance, even after the lapse of some years, the previously established rhinogenous origin could not be doubted at all.

The detrimental result of procrastination is vividly illustrated in the following data, compiled by Tibor Bajkay in 1937, from the Budapest Oto-Rhino-Laryngological Clinic. They operated upon 54 patients for retrobulbar neuritis, in 29 of whom there was a positive rhinological finding. In every one of these the posterior sinuses had been opened. Of those with positive rhinological findings, 5 fully recovered, 21 improved, and 3 remained uncured. Of the number of those with negative findings, 4 were cured, 15 improved, and 6 remained uncured. It was furthermore well established that patients who presented themselves 3 to 4 days after deterioration of the vision had set in, achieved full sight, whereas for those who presented themselves only after 8 to 14 days, the operation resulted in only an improvement. The nine patients whose condition was not improved by the operation,

** On the occasion of Onodi's demonstration I saw four such preparations in which the optic canal ran a course in one of the posterior sinuses to a length of 6 to 10 mm.

presented themselves for treatment after a lapse of two weeks.

Among their data a contralateral case was also described, in which a rightsided ethmoiditis gave rise to the occurrence of a retrobulbar neuritis on the left side and in two weeks, subsequent to the cleansing of the sinus in question, the eye healed with full vision. An anatomical preparation, corresponding to this case, was among the slides demonstrated by Onodi, which I had the pleasure of seeing.

S. R. Gifford in a relevant discussion writes: "In spite of the floods of ink that have been spilled on this question during the past 10 years, it is still decidedly a question and may be resolved into three principal parts: In what percentage of cases of retrobulbar neuritis is sinusitis the cause? Can retrobulbar neuritis be caused by the disease of the sinuses giving no evidence on rhinologic or roentgenologic examination; if so, what is the pathology of the disease of the sinuses? In the presence of retrobulbar neuritis with no apparent cause and with no nasal evidence of sinusitis should one advise exploratory operation on the sinuses?"

In regard to the first question I believe that the relevant statistics are not conclusive, because there is yet no consistent decision as to the rhinogenic origin of neuritises and therefore as many statistics are compiled as there are points of view upon which they are based. Suffice it to say that neuritis of rhinogenous origin is a rare disease, but that it does occur.

To the second question I can give an answer on the ground of my own cases. In every such case, when per exclusionem the origin of an optic neuritis can be located only in the accessory cavities, there is the possibility of a closed empyema even if the nasal and X-ray findings are negative. Therefore, in such instances —and this is the answer to the third question—the explorative opening of the posterior sinuses is imperatively necessary.

B. Serous iritis

In 1929, 10 cases of serous iritis came under my observation, and by chance I found that a nasal swab soaked in cocaine-adrenalin solution was apt to bring about a striking improvement. I dealt with this topic at length in an article which appeared in the Zeitschrift für Augenheilkunde in 1930. This publication did not provoke much comment, and for this reason I again call the attention of opthalmologists to this important fact. Prior to entering into the explanation of the pathogenesis, I shall describe various types from the cases I have observed.

Case 1. S. N., a female, aged 68 years, called on me on May 1, 1928, with the complaint that for one week her right eye had been inflamed.

Examination: There was an explicit serous iritis in the right eye; the fundus was invisible. The left eye was normal. A blood test proved negative.

Some weeks previously she had had a cold. The described ocular condition persisted with unvaried intensity for three weeks, associated with tormenting headaches, and at this time a mild iritis supervened also in the fellow eye, preceded, two days previously, by a neuritis. Because of the tendency of the pupils to dilate I dropped scopolamine into both eyes once a day only. Then I told the patient to consult a rhinologist, who established the presence of a chronic rhinitis and deviation of the septum. On the left side the nose proved normal. On the right side I applied cocaine-adrenalin swabs daily, whereupon the inflammatory phenomena as well as the precipitates, likewise the tormenting headache, showed a quick and striking improvement, and by July 8th both eyes had recovered with full vision. Meanwhile, when the precipitates disappeared and the fundus could be examined, a markedly developed optic neuritis could be observed in the primary eye also.

Comment: As will be seen from the description, in the primary eye a serous iritis made its appearance, followed later by an optic neuritis, and after the lapse of 25 days serous iritis supervened also in the other eye, having been preceded, by some days, by an optic neuritis. The fact that the condition of the primary eye in spite of the customary treatment gradually deteriorated until nasal treatment was begun,

and that from this time on a striking recovery was set in motion, and also that the torturing headache abated, authorized me to designate this bilateral iritis as of rhinogenous origin. For it is plain that the abatement of the severe symptoms, the elimination of the precipitates, and regression of the optic neuritis cannot by any means be attributed to the once daily administered scopolamine solution, but could be ascribed to the nasal treatment.

Case 2. H. E., a female, aged 22 years, had had a cold some weeks previous to her calling on me on the 14th of November, 1928.

Examination: Right eye normal; in the left eye a severe serous iritis.

Atropine was prescribed to be administered five times daily. The tuberculin test was negative; the blood Wassermann test positive.

Nasal finding on the 28th of November: rhinitis on the left side. These tests lasted for 14 days, during which time the condition of the eye, in spite of the energetic atropinization, gradually became worse, violent headaches supervened, and the precipitates greatly increased. Then, encouraged by the good results achieved in the first case, I resolved to try, instead of an antisyphilitic cure, treatment of the patient with only cocaine-adrenalin swabs. This resolution was prompted by an older statement by Römer, in which he said that even in case of positive blood test every iritis need not be regarded as syphilitic. The cocaine-adrenalin treatment was successful, inasmuch as after a few days the pains abated and the inflammatory phenomena of the eye considerably subsided, so that by the 28th of December I was able to examine the eyeground, which proved to be unimpaired; vision was 5/10. At this time the inflammatory symptoms were only moderate. In 10 days, however, the patient returned with the surprising statement that a few days before, the other eye also had become inflamed, while the condition of the primary eye was aggravated. Upon my interrogating her, she confessed to have neglected the nasal treatment for several days, in view of the improvement that had set in.

Examination: Well-developed serous iritis bilaterally with copious precipitates. Temperature 37.4°C. Nasal finding: The rhinitis present on the primary side displayed a vigorous recurrence. With adequate nasal treatment and with the administration of atropine both eyes recovered with full vision by the 9th of April. After the disappearance of the precipitates the eyeground examination, carried out meanwhile, showed both papillae to be intact.

Comment: From this description it is clear that in an individual, strongly syphilitic, a bilateral serous iritis supervened in an interval of several weeks, and in spite of the omission of antiluetic treatment, tamponade of the nose led to a complete cure. It is a striking circumstance that inflammation supervened in the other eye only when the patient, at the time when there was considerable improvement of the primary eye, neglected the nasal treatment—as superfluous—and in this way the rhinitis became exacerbated. As, however, the optic nerve remained unimpaired during the entire duration of the disease, I would conclude that in the present case the transmigration of the pathogenic agents was by way of the ciliary nerves.

Case 3. T. M., a girl of 15 years, suffered in September, 1928, from a bad cold in the nose with copious secretion. Some weeks later, when these symptoms had passed, the right eye began to be inflamed, and in three weeks the left eye also. She came to consult me on the 6th of December, until which time she had had no treatment whatever.

Examination: Well-developed serous iritis on both sides with abundant precipitates. Vision in the right eye was reduced to the ability to see hand movements, and in the left eye to the ability to count fingers at a distance of one meter. The girl was poorly developed, her skin pale. Twelve of her 13 brothers and sisters had died. The Wassermann test was positive.

Rhinological findings: Rhinitis chronica et sinusitis ethmoidalis on the right side. In the present case—for the sake of comparison—I entirely omitted the treatment of the nose and besides the administration of atropine I confined myself to applying antiluetic treatment. From the time of admission until the 11th of March she received 18 injections of bismuth and 8 of salvarsan. In spite of this the precipitates, forming a mass on both corneae, scarcely diminished, although the inflammatory symptoms of the eye subsided. As the antiluetic treatment, carried on for 90 days, proved to be of very little avail, I stopped it and instead commenced the tamponade of the nose, the more so as meanwhile epistaxes occurred. Upon the application of this method the dwindling of the precipitates was accelerated, so that by the 20th of April the vision in the right eye had risen to 5/20, of the left eye to 5/15. It could now be seen that both papillae were normal. The patient was obliged to leave for family reasons, taking with her adequate instructions for steam inhalations.*

Comment: As can be seen from this case the antiluetic treatment continued for 90 days influenced the healing of the eyes only negligibly, whereas a striking success followed nasal treatment continued for 35 days. It can, therefore,

* A pot of 300-400 c.c. capacity is filled with boiling camomile-tea solution, and covered with a paper funnel, the tip of which is fitted into the nostril.

be stated that serous iritis, even in the presence of a strongly positive Wassermann test, also may be of rhinogenous origin.

Case 4. B. L., a female, aged 35 years, consulted me on November 3, 1937, on account of dim vision in her left eye.

Examination: The right eye was normal; the left eye entirely devoid of inflammation, with bulbar conjunctiva white, the cornea sound. However, in the aqueous chamber, scarcely noticeable, tiny precipitates could be observed.

Diagnosis: Cyclitis. Upon inquiry, she asserted that during the last 15 years she had received antiluetic treatment on several occasions; and that now for one week she was again being treated. Under the circumstances I did not think of a rhinogenous origin. As, however, the eye became strongly injected on the next day, the precipitates increased, and the inflammatory symptoms of the iris displayed during the course of the next 10 days a gradual aggravation, I insisted on rhinoscopy. The presence of a subacute rhinitis of the left side was found. For this reason, I forthwith applied cocaine-adrenalin swabs, a treatment that was conducive to the rapid subsidence of all inflammatory signs and the striking disappearance of the precipitates, so that within the following 10 days complete recovery ensued. According to the statement of the patient, the effect of the tamponade was to cause an abundant thick discharge to be evacuated from that side, through the nostril as well as posteriorly, which she had failed to observe up to that time.

Comment: Serous iritis broke out while antileutic therapy was in course of being administered. It gradually became aggravated in spite of vigorous atropinization; but upon the introduction of nasal treatment full recovery was effected within a short time.

Case 5. E. P., a female, 20 years of age, consulted me on the 5th of May, 1932, complaining of an inflammation of the right eye of a week's standing. I found a severe serous iritis, with copious precipitates. The patient was pale, emaciated, and the internal and screening examinations established the presence of an apical catarrh. Rhinoscopy revealed the presence of a severe rhinitis on the right side. As an experiment I ordered nasal tamponades, on the assumption that perhaps the presence of tuberculosis does not exclude the nasal origin of serous iritis just as had been the case in the presence of syphilis. I was not disappointed, for upon the administration of nasal treatment all the phenomena of the inflammation disappeared at an astonishingly rapid rate, and the patient regained full vision within eight days, by the 13th of May.

Comment: As is evident from this case, serous iritis may be of rhinogenous origin also in the presence of tuberculosis and independent of the Koch bacillus. This I had declared to be possible as early as 1930.* My concept was corroborated by Löwenstein of Vienna (1936), who, on the basis of an experience of 35 years and of 34,000 examinations, made the statement that "the mere presence of the Koch bacillus does not necessarily indicate that it was the etiologic agent."

Case 6. A. K., a male, 24 years of age, was admitted on the 17th of November, 1937, complaining that his left eye had been inflamed for a few days and that previous to this he had suffered from rhinitis.

Examination: There was serous iritis of the left eye in the absence of lues and tuberculosis.

Rhinoscopy: A chronic rhinitis of the left side was found. Atropine and a nasal tamponade were administered, with but moderate improvement. By the 22d of December he had recovered completely. As soon as the precipitates had sufficiently disappeared, I examined him with the ophthalmoscope and to my surprise observed a moderate optic neuritis, which afterwards gradually regressed.

Comment: The importance of this case lies in the fact that only one eye was affected and yet optic neuritis appeared, which later wholly disappeared with the outstanding symptoms of iritis. It might be concluded that nasal treatment administered in time gradually stopped the reserves of the pathogenic agents migrating from the sinuses into the uvea. Thus the iritis extending to all three parts of the uvea began to regress even prior to the ascending neuritis having extended to the other eye through the lamina cribrosa. This is what took place in case 1, in which I began the nasal treatment only on the twenty-third day, when the fellow eye had become inflamed. Thus I observed —for the second time within 10 years—an ascending optic neuritis of the primary eye in connection with iritis.

It is therefore desirable that every serous iritis be subjected to ophthalmoscopic observation, and if this were done, perhaps my relevant observation will not continue to remain isolated in the literature.

On the other hand, the results in this sixth case indisputably point to the analogy between serous iritis and sympathetic ophthalmia.

I stated this analogy in full in 1937** and in order to disperse doubts I shall supplementarily report further data which I have recently discovered in the literature, and which I had not yet utilized in

* Zeitschrift für Augenheilkunde, 1930, and Archiv für Augenheilkunde, 1935.

** American Journal of Ophthalmology, 1937, v. 20, p. 618.

my writings. In chronological order A. v. Graefe was the first to give expression to the assumption that in a bilateral spontaneous uveitis the disease of the secondary eye supervenes under the influence of the primary eye.

Professor Schnablel, Vienna, 1902, one of the most prominent ophthalmologists of his time, who analyzed the pending questions of pathogenesis with an impartial and irresistible logic, rigidly adhered to the assumption that spontaneous uveitis and sympathetic ophthalmia are identical and denied the exclusive ability of injuries to bring about the latter disease. According to him sympathetic iridocyclitis as well as spontaneous iridocyclitis are produced by the same pathologic agent and in both of the latter this originates somewhere in the human body and reaches the uvea by the same route in both of these pathologic forms.

Elschnig, 1910, formulated his opinion on this subject, as described by Peters, as follows: ". . . and as regards the relation of sympathetic ophthalmia to spontaneous iridocyclitis, in this matter Elschnig repeatedly accentuated the identity. The simultaneous morbidity of the two organs is an evidence of its constitutional origin. If the two eyes succumb with a certain interval between, the inflammation of the secondary eye originates from the primary eye."

Finally, Meller in 1915 wrote as follows: "Idiopathic* uveitis is the same pathological form which we know of old as sympathetic ophthalmia. . . . Therefore, we should rather refrain from adhering too much to the term sympathetic ophthalmia, which we have failed to substitute with another term, up to the present, because without knowing the pathologic agent this is aimless."

After these preliminaries I hope it will cause no special inconvenience to the reader, if in dealing with the pathogenesis in one case I resort to the term "spontaneous iritis" and in another to "sympathetic ophthalmia," these two pathologic forms being identical; the difference between the two is only as to their etiology.

Pathogenesis

Serous iritis is an affection of a much greater importance than optic neuritis, on the one hand because it can be observed in a much greater number of cases than can the latter, on the other hand because it is often bilateral and therefore the neglect of adequate therapy is liable to result in a serious disability.

Ten years have passed since the publication of my first treatise, during the course of which I have had numerous cases of serous iritis under my care, and the conviction has grown upon me that this is a wholly separate affection that is caused by a specific agent and is independent of syphilis as well as of tuberculosis. In every case, two weeks prior to the affection of the eye, a catarrhal infection had occurred which ran a mild course and was confined chiefly to the upper respiratory passages, the patient being bedridden for only a day or two, if at all.

I was able as well to establish that in certain years only 1 to 2 cases occurred while in others 10 to 15, usually when a mild endemic influenza was prevalent in our city. My serous-iritis cases presented themselves, without exception, in the season October to April; accordingly I would call it a *seasonal disease*.** In this respect my opinion gained fresh reinforcement in the data of Broman Tore, according to whom 120 violent iritises

* The term idiopathic was used by Schirmer for those spontaneous iritises, the etiology of which is unknown.

** See American Journal of Ophthalmology, 1937, v. 20, 618.

occurred after 650 cataract operations (17 percent), making their appearance for the most part during the winter.

The excellent results achieved with nasal tamponade justify me in regarding serous iritis as *of rhinogenous origin.*

For this reason we should search for the causative agent in the accessory cavities. It is the influenza bacillus that is most often found in the accessory cavities, and due to its peculiarities it is apt to bring about serous iritis, as I showed in 1937.

Besides the Pfeiffer bacillus the Diplococcus catarrhalis has recently been brought to my attention.

In a publication from the Budapest First Surgical Clinic there appeared in 1938 a discussion of the bacteriology of the cavity of Highmore. From data derived from 50 cases, Dr. Szende and Dr. Murányi elaborated the findings in 26 chronic and 24 acute cases. In the discharge of the patients with chronic inflammations chiefly streptococci and staphylococci were found, while in the acute cases the discharge contained exclusively pneumococci and catarrhal diplococci in about equal proportions. According to these authors the greater part of their cases were of infections of influenzal origin. As, however, the openings of the antrum and of the ethmoidal sinus are in the same nasal passage, I am justified in presuming that these causative agents may be present in the latter also.

The pneumococcus cannot come into consideration, for, as is well known, this organism is apt to provoke much more serious changes in the eye than are seen in the manifestations of serous iritis. We must therefore turn our attention to the catarrhal diplococcus. This organism can be found on the mucosa of the nasopharynx even in normal conditions and according to the findings of the present writer it alone may be the causative agent of an inflammation of the cavity of Highmore. Although its pathogenicity is slight, it is nevertheless capable of bringing about a catarrh of the upper respiratory passages. O. Seifert found it in the discharge of patients with a light bronchitis while R. Pfeiffer cultivated it from the deeper respiratory ducts as the causative agent of puerile bronchopneumonias associated with influenza bacillus. Thus the Diplococcus catarrhalis may come into consideration as bringing about serous iritis, either alone or associated with the Pfeiffer bacillus.

The question still remains as to how the causative agents reach the eyeball from the sinuses. In my opinion this can take place in two ways; namely, either by the hematogenous route, or by direct passage. The possibility of the former is verified by the known circulatory conditions; as to the latter the pathologic-histological findings offer sufficient evidence.

As I have previously mentioned, in the numerous cases of serous iritis observed by me the rhinological examination established, without exception, the acute catarrhal affection of the mucosa, or its exacerbation.

According to Hajek, when the sinuses succumb to acute catarrh, the mucosa, in its entire thickness, down to the bony wall is affected with an inflammatory edema and swells to three times its normal thickness. Within this, numerous smaller and larger cavities form which become filled with clotted serum, and the mucosa contains polynuclear leucocytes and lymphocytes.

According to this author a high degree of edematous inflammation in the presence of a relatively mild acute inflammation is a specific characteristic of the mucosa of the ethmoidal sinus. As a result, the inflammation extends relatively easily and speedily to the deepest layers of the mucosa, which acts as a periosteum.

In this sense, in the catarrhal affections of the sinuses (in contrast with empyema) no defensive dam of inflammation arises, and the lymphocytic infiltration can extend unchecked to the bony wall. It will not be hindered in its progress here either, for according to the previously mentioned discovery of Herzog, the medullary cavities of the bony wall provide a straight path for the etiologic agents toward the optic canal. Here it is easy for them to get into the pia mater and thence to settle down in the uvea.

It is thus readily believable that one of the chief factors in the cure of serous iritis is the treating of the nose, for when we insure an undisturbed drainage of the pathologic discharge of the sinus, we at the same time put an end also to the reserve of migrating causative agents. The resisting power of the attacked eye, supported by mydriatics, enables it to eliminate the remaining pathologic agents and recovery follows.

Hajek further writes: "That the rapid drainage of the discharge from a cavity is the most important requirement for a prompt recovery, is a universally valid basic rule of pathology."

Iritis serosa is in most cases unilateral, and the affection only infrequently makes its appearance on the other side also. This happens either some weeks later or may ensue after the primary eye has become blind, after a period of years. In the latter case Weigelin observed, subsequent to the removal of the primary eye, considerable improvement in the secondary eye, and the histological examination revealed a fresh inflammatory focus in the uvea, just as is the rule in sympathetic ophthalmia.

Serous iritis occurs more frequently than optic neuritis because the former, unlike optic neuritis, may supervene even in the presence of the thickest bony wall. In spite of this, just as in the case of the latter, it is of less frequent occurrence than is the inflammation of the accessory cavities. The cause lies in the fact that for the development of serous iritis in addition to a catarrhal sinusitis another factor is also necessary—the condition that may develop in the uvea (in addition to constitutional factors) from an injury, or an unsuccessful operation, or an intraocular tumor.

According to the statement of other writers retrobulbar neuritis does not occur in juveniles, and I can allege the same as to serous iritis, for amongst the numerous patients under my observation there was none under 14 years of age. This means that both pathologic forms alike are related to the diseases of the posterior sinuses, which are not developed before the age of puberty. On this basis White's statement that optic neuritis is caused by disease of the antrum is in error, for this cavity has been shown to be present even in infants.

Summary

As can be seen from the foregoing, the pathogenesis of optic neuritis and of serous iritis displays considerable differences; they have, however, many points in common.

The optic neuritis of rhinogenic origin is the outcome of an *aseptic process* going on in the optic canal, and is brought about exclusively by mechanical factors. Its origin is in the empyema of the posterior sinuses, or the chronic hypertrophic inflammation of the latter.

Spontaneous serous iritis, even in the presence of syphilis or tuberculosis, is in every instance the outcome of a *catarrhal infection.*

The source of infection is the catarrhal inflammation of the posterior sinuses, and pathogenic agents migrate directly from the sinus into the canal and thence, by way of the pia mater, into the uvea.

INTRAOCULAR TENSION IN ELECTROPYREXIA*

A PRELIMINARY REPORT

H. C. ERNSTING, M.D.
Cleveland, Ohio

There has been a most marked interest in electropyrexia within the past few years, but published data dealing with the effects of electropyrexia on the intraocular tension have not come to this writer's attention.

It was thought quite possible that an elevation of the intraocular tension might occur in electropyrexia and hence prove a contraindication to the treatment of those eye diseases (such as corneal ulceration, with the possibility of herniation or perforation of the cornea and glaucoma) in which such an event might prove disastrous. This study was therefore undertaken in an attempt to determine the effects of this type of artificially induced fever on the intraocular tension.

Neither the state of refraction nor the age has any appreciable effect on the intraocular tension. Holocaine has no influence on the intraocular pressure, though it does affect the cornea by producing "polygonal dry spots" which quickly disappear.[1] There are, however, many factors that may affect the intraocular tension, some of which are: certain drugs, external influences, action of the sympathetic and trigeminal nerves, action of the muscles, and so on.[2]

In electropyrexia or hyperpyrexia as induced by the Kettering Cabinet, it was found in the majority of 19 cases studied that the intraocular tension was definitely affected. The patients who were examined were suffering from various types of arthritis and only two presented ocular pathology of any significance.

The procedure, prior to fever therapy, varied with the case to be treated. During fever therapy a sedative was given shortly after the temperature began to rise. The majority of patients received morphine gr. 0.25; some few received equivalent doses of pantopon or codeine, and an occasional patient received sodium amytal gr. 3. The dose was repeated, if the treatment was long or the patient became restless. The fluid level was maintained by the oral administration of 0.6 percent saline or, on rare occasions, of plain water. The average patient took between 2,000 and 2,500 c.c. of fluid per treatment. The rectal temperature was watched closely. Ice applied to the face and a fan situated near the head contributed somewhat to the patient's comfort. The duration of each treatment varied with the individual case.

Most investigators believe that the effects of artificial fever are unlike those of natural fever, but there are a few[3] who report the effects to be quite similar. It is almost universally believed, however, that fever stimulates the development of immune bodies, favors phagocytosis, exerts an unfavorable influence upon the growth of certain bacteria, and diminishes the potency of toxins. Following a fever treatment, it is stated that the white blood cells are constantly increased. The polymorphonuclear leukocytes show a marked increase[4] and there is a definite shift to the left in the Schilling hemogram. The red blood cells and hemoglobin do not show much change. There is a loss of

* From the service of Drs. P. G. Moore, G. L. Miller, and B. J. Wolpaw, Department of Ophthalmology of the Western Reserve University School of Medicine and the Cleveland City Hospital.

TABLE 1

INTRAOCULAR TENSION IN ELECTROPYREXIA

Case	Disease	Hours per Treatment	Temperature	Blood Pressure		Intraocular Tension		
				Before	After	Before	During	After
			Fahrenheit	mm. Hg	mm. Hg	mm. Hg	mm. Hg	mm. Hg.
M. S.	Gonorrheal arthritis	8	106–107	138/100	104/70	20 18	20 18	17 O.D. 16 O.S.
W. T.	Tabes arthritis	8	105–106	140/100	60/60	20 17	20 17	15 O.D. 14 O.S.
T. H.	Infectious arthritis	5	106–107	110/70	80/0	20 18	20 18	15 O.D. 15 O.S.
T. H.	Infectious arthritis	6	106–107	130/90	120/80	20 18	20 18	17 O.D. 15 O.S.
L. N.	Infectious arthritis	3	104–105	120/68	120/70	18 20	18 20	18 O.D. 20 O.S.
H. D.	Interstitial keratitis	5	105–106	120/74	110/70	18 18	18 18	14 O.D. 20 O.S.
F. D.	Atrophic arthritis	3	103–104	112/60	92/58	20 20	20 20	14 O.D. 14 O.S.
A. A.	Atrophic arthritis	3	103–104	168/70	130/60	23 23	23 23	15 O.D. 15 O.S.
R. T.	Gonorrheal arthritis	8	106–107	136/100	104/60	15 20	15 20	12 O.D. 15 O.S.
M. H.	Infectious arthritis	4	105–106	138/90	110/80	23 23	22 23	20 O.D. 20 O.S.
R. D.	Atrophic arthritis	3	103–104	126/80	114/70	23 23		15 O.D. 15 O.S.
L. F.	Arthritis iritis	4	105–106	142/98	120/74	20 20		18 O.D. 18 O.S.
K. S.	Infectious arthritis	6	106–107	108/78	100/80	24 26		15 O.D. 17 O.S.
F. M.	Gonorrheal arthritis	5	105–106	140/76	130/70	17 18		15 O.D. 16 O.S.
M. P.	Atrophic arthritis	3	103–104	124/78	110/70	20 20		18 O.D. 18 O.S.
E. T.	Spondylitis	3	104–105	118/62	88/58	15 15		13 O.D. 13 O.S.
M. F.	Atrophic arthritis	3	103–104	122/80	120/68	20 20		20 O.D. 20 O.S.
R. S.	Osteomyelitis of hip	4	101–102	120/70	98/60	17 17		15 O.D. 15 O.S.
D. F.	Atrophic arthritis	3	103–104	129/70	92/54	22 16		17 O.D. 15 O.S.

blood and tissue chlorides and of carbonic acid, and in patients who are subjected to prolonged treatment at high temperatures this loss of acid ions induces some degree of alkalosis.[5] This alkalosis may be controlled by the oral administration of 0.6-

percent saline solution and the inhalation of oxygen-carbon dioxide. The blood sugar, nonnitrogenous phosphorous, and serum calcium show little if any change. At the onset of treatment the systolic blood pressure may be elevated slightly, but after treatment it is lowered. During the maintenance period the basal metabolic rate is reported[5] to be increased 5.5 percent for each degree (Fahrenheit) of temperature elevation. A marked increase in the number and size of the visible capillaries and the rate of flow has been reported, and it is said that this may be taken as a practical index of the circulatory changes that occur throughout the entire body during fever treatment.[3]

One would expect similar changes to occur in the eye, and the eye does present changes that may be attributed to the effects of hyperpyrexia. The conjunctiva becomes injected, and it is a question whether the fundus does not appear slightly more vascular. The intraocular tension, which was determined under holocaine anesthesia and by means of the Schiötz tonometer, showed little or no change in 10 cases that were examined during treatment, but the tension, which was taken under the same conditions, 30 minutes after treatment showed a definite drop in the majority of the 19 cases examined (table 1).

Conclusion

No conclusions may be drawn from a preliminary report of this type. It is known that the intraocular tension generally follows passively the changes in general blood pressure. It is further known that the blood pressure may be definitely influenced by changes in the capillary system. In the majority of the 19 cases that were examined 30 minutes after fever treatment, the intraocular tension was lowered. It is evident that further study and investigation will be necessary.

I wish to express sincere thanks to the Department of Arthritis and Fever Therapy, whose courteous coöperation has permitted this work.

References

[1] deSchweinitz, G. Diseases of the eye. Philadelphia, W. B. Saunders Co., 1924, p. 93.
[2] Fuchs-Duane. Textbook of ophthalmology. Ed. 8, New York, J. B. Lippincott Co., 1924, pp. 80-83.
[3] Shepley, E. E. Fever therapy. The Canadian Med. Assoc. Jour., 1937, v. 37, p. 341.
[4] Krusen, F. H. Studies of the blood picture before and after fever therapy. Abstracts of papers and discussions, Fifth Annual Fever Conference, Dayton, Ohio, May, 1935, pp. 91-96.
———. Present status of fever therapy produced by physical means. Jour. Amer. Med. Assoc., 1936, v. 107, Oct. 10, p. 1215.
[5] Simpson, W. M. Studies on the physiology of fever. Jour. Amer. Med. Assoc., 1936, v. 106, Jan. 18, p. 246.
Parsons. Diseases of the eye. New York, The Macmillan Co., 1931, pp. 14-19.
Lepore, M. J. Chloride and water metabolism in patients with artificial fever. Abstracts of papers and discussions, Fifth Annual Fever Conference, Dayton, Ohio, May, 1935, pp. 38-42.
Hargraves, M. M., and Doan, C. A. The physiologic response of the hemopoietic tissues to artificially induced fever. Abstracts of papers and discussions, Fifth Annual Fever Conference, Dayton, Ohio, May, 1935, pp. 51-61.
Solomon, H. C., and Kopp, I. Fever therapy. New England Med. Jour., 1937, v. 217, Nov. 18, pp. 805-815.
Sittler, E. C. The Kettering hypertherm. Abstracts of papers and discussions, Fifth Annual Fever Conference, Dayton, Ohio, May, 1935, pp. 115-117.

CAUSES OF BLINDNESS IN INDIANA

D. H. Row, M.D.* and C. D. Chadwick**
Indianapolis, Indiana

The following report, based on the physical ocular examinations of 2,657 applicants for blind pension in the state of Indiana, gives a fair insight into the common causes of blindness in the mid-western part of the United States. The examinations were made by physicians practicing in or near the county in which the applicant resided, and the examiner was either an oculist or an eye, ear, nose, and throat specialist, as required by the Welfare Act of Indiana for 1936.

As a standard qualifying requirement, the central vision of the better eye of the applicant had to be reduced to 20/200 or less. All of the individuals included in this analysis were so handicapped. All applicants were adults. However, not all applicants were eligible for blind pension because of other considerations; usually financial support from other sources.

Through the coöperation of the National Society for the Prevention of Blindness, a standard form of the causes of blindness, prepared by the Committee on Statistics of the Blind, was obtained. Proceeding with this chart, which has a detailed cross-classification of topographical listings—such as, glaucoma, cataract, iritis, and corneal opacity—along its upper border, and etiological factors—such as, ophthalmia neonatorum, syphilis, trachoma, and diabetes—on the side margin, a topographical and an etiological factor was listed on each of the 2,657 individual records. After this was accomplished, it was a relatively simple matter to enter the totals on the chart.

The analysis of these records is, for obvious reasons, inaccurate; for example, many of the applicants have been blind for years with secondary changes, such as cataract, developing after blindness, obscuring posterior disease. History of the applicant's loss of vision, in many cases, only confused the issue because of the usual lack of knowledge on the part of the applicant as to the real cause of his blindness.

Cases reported on the individual applicant's forms as cataract and glaucoma, were classified as glaucoma, since it seemed reasonable to consider the more profound and serious disease as the actual cause of blindness. Likewise, cases reported as senile cataract, but in which there was no light perception, were classified as posterior disease, without reference to the cataract.

Some applicants had lost one eye from injury or disease years before the loss of the second eye. Unless the question of sympathetic ophthalmia was involved, the loss of the first eye was not considered in compiling these figures on blindness.

Indefinite etiological classifications—such as, "probably syphilis"—were classified as "undetermined by physician," so that the incidence of syphilis in the final totals is probably underestimated.

Sympathetic ophthalmia was classified as uveitis, since the chart used had no listing for sympathetic ophthalmia. This is probably not so serious a deficiency of the record as is, at first consideration, imagined, for the factor of *post hoc propter hoc* comes into prominence when considering this disease, and without

* Consulting Ophthalmologist for Indiana Board of Industrial Aid for the Blind.

** Executive Secretary of Board of Industrial Aid for the Blind of the Indiana Public Welfare Department.

microscopic examination or even clinical observation of the active process of the sympathetic disease the diagnosis is often questionable.

Of all the etiological factors listed, probably the most accurate is trachoma, since the disease and its scar-tissue sequelae are superficial and quite characteristic.

Due to the frequent use of the vague term "senility" as an etiological factor, this was added to the chart as the only deviation from the printed form of the Committee on Statistics of the Blind.

Because of the large and cumbersome nature of the original cross-classification chart, it has not been included with this report. The following two charts are self-explanatory. Chart 1 gives the numerical frequency of the local eye disease. Chart 2 gives the numerical frequency of the underlying etiology of the eye disease.

CHART 1

TOPOGRAPHICAL INCIDENCE

	Cases	Pathology
1.	20% 544	LENS OPACITY (cataract)
	363	Senility
	56	Congenital and hereditary, not specified
	45	Undetermined by physician
	24	Diabetes
	8	Hereditary and familial
	7	Trachoma
	5	Etiology, not specified
	4	Vascular diseases
	4	Infections, specified
	3	Nephritis and other kidney diseases
	3	Systemic diseases, not specified
	2	Measles
	2	Syphilis, prenatal
	2	Syphilis, not specified
	2	Tuberculosis
	2	Injuries incidental to surgery
	2	Toxic poison, specified
	2	Toxic poison, not specified
	1	Ophthalmia neonatorum
	1	Firearms
	1	Other explosives, specified
	1	Household activities
	1	Trauma (including burns)
	1	Injuries, not specified
	1	Systemic diseases, specified
	1	Unknown to science
2.	14% 380	OPTIC ATROPHY
	173	Etiology undetermined by physician
	58	Syphilis, not specified
	16	Meningitis
	14	Neoplasms
	12	Congenital and hereditary, not specified
	11	Vascular diseases
	9	Senility
	8	Infections, specified
	7	Toxic poison, not specified
	6	Syphilis, acquired
	7	Nonindustrial injuries, specified
	6	Toxic poison, specified
	5	Nephritis and other kidney diseases
	5	Systemic diseases, specified
	4	Firearms
	4	Injuries, not specified
	4	Diabetes
	3	Measles
	3	Typhoid
	3	Trauma (including burns)
	2	Syphilis, prenatal
	2	Trachoma
	2	Explosives, specified
	2	Street and traffic accidents
	2	Birth injuries
	2	Alcohol (methyl, wood, denatured, etc.)
	2	Systemic diseases, not specified
	2	Etiology, not specified
	1	Gonorrhea (excluding O.N.)
	1	Scarlet fever
	1	Smallpox
	1	Industrial diseases (including poison)
	1	Tobacco
	1	Congenital and hereditary, prenatal
	1	Noninfectious diseases, central nervous system
3.	14% 380	HYPERTENSION (glaucoma)
	294	Unknown to science
	21	Undetermined by physician
	16	Vascular disease
	13	Congenital and hereditary, not specified

CHART 1 *(continued)*

			Cases	Pathology
			5	Infections, specified
			3	Toxic poison, specified
			3	Nephritis and other kidney diseases
			3	Hereditary and familial
			3	Senility
			2	Infections, not specified
			2	Explosives, specified
			2	Toxic poison, not specified
			2	Diabetes
			1	Measles
			1	Smallpox
			1	Syphilis, not specified
			1	Street and traffic accidents
			1	Injuries incidental to surgery
			1	Trauma, including burns
			1	Injuries, not specified
			1	Neoplasms
			1	Diseases of pregnancy and childbirth
			1	Systemic diseases, specified
			1	Etiology not specified
4.	13%	346		ULCERATIVE KERATITIS
			194	Trachoma
			34	Undetermined by physician
			30	Ophthalmia neonatorum, not specified
			18	Smallpox
			16	Infections, not specified
			10	Trauma, including burns
			7	Nonindustrial injuries, specified
			6	O.N., gonorrheal
			5	Gonorrhea (excluding O.N.)
			5	Infections, specified
			4	Injuries, not specified
			3	Measles
			2	Tuberculosis
			2	Explosives, not specified
			2	Congenital and hereditary, not specified
			1	Meningitis
			1	Scarlet fever
			1	Syphilis, not specified
			1	Explosives, specified
			1	Toxic poison, specified
			1	Vascular diseases
			1	Systemic diseases, specified
			1	Etiology not specified
5.	5%	135		DISORGANIZED, ATROPHIC, PHTHISIC EYEBALL
			19	Etiology, undetermined by physician
			13	Firearms
			12	Injuries, not specified
			11	Ophthalmia neonatorum, not specified
			11	Infections, not specified
			9	Infections, specified
			5	Measles
			5	Trachoma
			5	Explosives, specified
			5	Trauma (including burns)
			5	Congenital and hereditary, not specified
			4	Meningitis
			4	Scarlet fever
			4	Nonindustrial injuries, specified
			3	Toxic poison, specified
			2	Gonorrhea (excluding O.N.)
			2	Ophthalmia neonatorum gonorrheal
			2	Smallpox
			2	Tuberculosis
			2	Typhoid
			2	Household activities
			2	Injuries incidental to surgery
			1	Ophthalmia neonatorum
			1	Syphilis, not specified
			1	Street and traffic accidents
			1	Toxic poison, not specified
			1	Neoplasms
			1	Etiology not specified
6.	3%	77		UVEITIS
			27	Etiology undetermined by physician
			17	Injuries, not specified
			6	Trauma (including burns)
			4	Nonindustrial injuries, specified
			3	Tuberculosis
			3	Infections, specified
			3	Infections, not specified
			2	Syphilis, prenatal
			1	Meningitis
			1	Smallpox
			1	Syphilis, acquired
			1	Syphilis, not specified
			1	Firearms
			1	Explosives, specified
			1	Explosives, not specified
			1	Household activities
			1	Toxic poison, specified
			1	Nephritis and other kidney diseases
			1	Etiology not specified
			1	Senility
7.	3%	71		RETINAL DEGENERATION, INCLUDING RETINITIS PIGMENTOSA
			15	Hereditary and familial
			15	Congenital and hereditary, not specified

CHART 1 *(continued)*

		Cases	Pathology
		14	Etiology undetermined by physician
		9	Senility
		6	Vascular diseases
		6	Etiology unknown to science
		1	Meningitis
		1	Syphilis, prenatal
		1	Toxic poison, not specified
		1	Diabetes
		1	Nephritis and other kidney diseases
		1	Congenital and hereditary consanguinity
8.	2.3%	65	RETINITIS
		15	Diabetes
		13	Nephritis and other kidney diseases
		11	Etiology undetermined by physician
		5	Syphilis, not specified
		4	Vascular diseases
		4	Congenital and hereditary, not specified
		4	Senility
		2	Toxic poison, not specified
		1	Measles
		1	Syphilis, prenatal
		1	Syphilis, acquired
		1	Infections, specified
		1	Explosives, specified
		1	Systemic diseases, specified
		1	Hereditary and familial
9.	2%	61	CHOROIDITIS
		29	Etiology undetermined by physician
		4	Vascular diseases
		3	Infections, not specified
		2	Measles
		2	Syphilis, not specified
		2	Toxic poison, specified
		2	Toxic poison, not specified
		2	Anemia and other blood diseases
		2	Systemic diseases, not specified
		2	Congenital and hereditary, not specified
		2	Senility
		1	Scarlet fever
		1	Syphilis, prenatal
		1	Tuberculosis
		1	Typhoid
		1	Explosives, specified
		1	Explosives, not specified
		1	Injuries
		1	Nephritis and other kidney diseases
		1	Hereditary and familial
10.	2%	51	KERATITIS, not specified
		20	Etiology undetermined by physician
		4	Infections, not specified
		3	Infections, specified
		2	Scarlet fever
		2	Explosives, specified
		2	Nonindustrial injuries, specified
		2	Trauma (including burns)
		2	Toxic poison, specified
		1	Measles
		1	Meningitis
		1	Syphilis, not specified
		1	Trachoma
		1	Tuberculosis
		1	Typhoid
		1	Firearms
		1	Explosives, not specified
		1	Street and traffic accidents
		1	Injuries incidental to surgery
		1	Injuries, not specified
		1	Toxic poison, not specified
		1	Hereditary and familial
		1	Congenital and hereditary, not specified
11.	2%	48	MYOPIA
		15	Etiology unknown to science
		13	Congenital and hereditary, not specified
		11	Etiology undetermined by physician
		4	Hereditary and familial
		1	Measles
		1	Syphilis, prenatal
		1	Syphilis, not specified
		1	Toxic poison, specified
		1	Systemic diseases, not specified
12.	2%	46	IRITIS
		20	Etiology undetermined by physician
		4	Trachoma
		4	Infections, specified
		3	Toxic poison, specified
		2	Injuries incidental to surgery
		2	Injuries, not specified
		2	Toxic poison, not specified
		2	Systemic diseases, specified
		1	Gonorrhea (excluding O.N.)
		1	Measles
		1	Syphilis, prenatal
		1	Tuberculosis
		1	Infections, not specified
		1	Explosives, specified

CHART 1 *(continued)*

		Cases	Pathology
		1	Congenital and hereditary, not specified
13.	2%	42	DISSEMINATED CHORIORETINITIS
		12	Etiology undetermined by physician
		4	Syphilis, not specified
		4	Hereditary and familial
		3	Syphilis, prenatal
		2	Infections, specified
		2	Anemia and other blood diseases
		2	Nephritis and other kidney diseases
		2	Vascular diseases
		2	Systemic diseases, specified
		1	Meningitis
		1	Scarlet fever
		1	Tuberculosis
		1	Typhoid
		1	Infections, not specified
		1	Injuries, not specified
		1	Toxic poison, specified
		1	Congenital and hereditary, not specified
		1	Senility
14.	1%	40	AMBLYOPIA, undefined
		21	Etiology undetermined by physician
		5	Congenital and hereditary, not specified
		2	Ophthalmia neonatorum, not specified
		2	Trachoma
		2	Vascular diseases
		1	Meningitis
		1	Ophthalmia neonatorium, gonorrheal
		1	Syphilis, not specified
		1	Infections, specified
		1	Infections, not specified
		1	Nonindustrial injuries, specified
		1	Trauma (including burns)
		1	Tobacco
15.	1.2%	37	IRIDOCYCLITIS
		15	Etiology undetermined by physician
		4	Injuries, not specified
		2	Infections, not specified
		2	Nonindustrial injuries, specified
		2	Systemic diseases, not specified
		2	Etiology not specified
		1	Meningitis
		1	Ophthalmia neonatorum, gonorrheal
		1	Trachoma
		1	Firearms
		1	Play or sport
		1	Street and traffic accidents
		1	Injuries incidental to surgery
		1	Trauma (including burns)
		1	Toxic poison, not specified
		1	Hereditary and familial
16.	1.2%	36	LESIONS, not specified
		10	Etiology undetermined by physician
		4	Trachoma
		4	Injuries not specified
		4	Congenital and hereditary, not specified
		3	Syphilis, prenatal
		2	Senility
		1	Measles
		1	Infections, specified
		1	Infections, not specified
		1	Firearms
		1	Household activities
		1	Birth injuries
		1	Nonindustrial injuries, specified
		1	Vascular diseases
		1	Systemic diseases, not specified
17.	1.2%	35	INTERSTITIAL KERATITIS
		16	Syphilis, prenatal
		6	Etiology undetermined by physician
		3	Syphilis, not specified
		2	Trachoma
		2	Tuberculosis
		2	Infections, not specified
		1	Typhoid
		1	Scarlet fever
		1	Toxic poison, specified
		1	Diabetes
18.	1%	27	ILL-DEFINED LESIONS, specified
		16	Etiology undetermined by physician
		2	Nephritis and other kidney diseases
		1	Syphilis, prenatal
		1	Infections, specified
		1	Infections, not specified
		1	Nonindustrial injuries, specified
		1	Trauma (including burns)
		1	Injuries, not specified
		1	Toxic poison, specified
		1	Toxic poison, not specified
		1	Vascular diseases
19.	1%	27	OPTIC NEURITIS
		4	Syphilis, not specified
		4	Toxic poison, not specified

CHART 1 *(continued)*

		Cases	Pathology
		4	Etiology undetermined by physician
		2	Meningitis
		2	Neoplasms
		2	Nephritis and other kidney disorders
		2	Vascular diseases
		1	Syphilis, prenatal
		1	Nonindustrial injuries, specified
		1	Injuries, not specified
		1	Alcohol (mythyl, wood, denatured, etc.)
		1	Toxic poison, specified
		1	Systemic diseases, specified
		1	Etiology not specified
20.	1%	22	DISORDERS OF OPTIC NERVE, not specified
		12	Etiology undetermined by physician
		5	Senility
		2	Diabetes
		1	Syphilis, acquired
		1	Injuries, not specified
		1	Nephritis and other kidney diseases
21.	1%	22	RETINAL HEMORRHAGE
		7	Vascular diseases
		4	Etiology undetermined by physician
		3	Nephritis and other kidney disorders
		2	Tuberculosis
		2	Diabetes
		2	Senility
		1	Syphilis, not specified
		1	Injuries, not specified
22.	1%	21	DETACHED RETINA
		9	Etiology undetermined by physician
		2	Injuries incidental to surgery
		2	Systemic diseases, specified
		2	Etiology unknown to science
		1	Infections, specified
		1	Firearms
		1	Household activities
		1	Trauma (including burns)
		1	Congenital and hereditary, not specified
		1	Senility
23.	.7%	19	DISORDERS OF THE EYEBALL, specified
		6	Etiology undetermined by physician
		3	Injuries, not specified
		2	Firearms
		2	Other explosives, specified
		2	Nonindustrial injuries, specified
		1	Syphilis, prenatal
		1	Explosives, not specified
		1	Neoplasms
		1	Etiology not specified
24.	.6%	16	PANOPHTHALMITIS AND ENDOPHTHALMITIS
		7	Infections, not specified
		3	Injuries, not specified
		1	Meningitis
		1	Syphilis, prenatal
		1	Trachoma
		1	Explosives, not specified
		1	Toxic poisons, specified
		1	Etiology undetermined by physician
25.	.5%	13	NEURORETINITIS
		3	Syphilis, prenatal
		3	Etiology undetermined by physician
		2	Congenital and hereditary, not specified
		1	Infections, specified
		1	Infections, not specified
		1	Toxic poison, not specified
		1	Vascular diseases
		1	Hereditary and familial
26.	.3%	10	DISORDERS OF THE EYEBALL, not specified
		4	Etiology undetermined by physician
		1	Ophthalmia neonatorum, not specified
		1	Infections specified
		1	Infections not specified
		1	Firearms
		1	Industrial injuries, not specified
		1	Etiology, not specified
27.	.3%	9	OPACITIES
		2	Infections, specified
		1	Trachoma
		1	Explosives, specified
		1	Street and traffic accidents
		1	Vascular diseases
		1	Systemic diseases, not specified
		1	Congenital and hereditary, not specified
		1	Etiology undetermined by physician

CHART 1 *(continued)*

	Cases	Pathology
28. .3%	9	DISLOCATED LENS
	5	Congenital and hereditary, not specified
	2	Etiology undetermined by physician
	1	Injuries, not specified
	1	Hereditary and familial
29. .3%	8	DISORDERS OF CHOROID AND RETINA, not specified
	4	Senility
	2	Etiology undetermined by physician
	1	Syphilis, not specified
	1	Etiology not specified
30. .2%	7	DISORDERS OF CHOROID AND RETINA, specified
	4	Etiology undetermined by physician
	1	Neoplasms
	1	Diseases of pregnancy and childbirth
	1	Etiology unknown to science
31. .2%	7	DISORDERS OF IRIS AND CILIARY BODY, specified
	3	Congenital and hereditary, not specified
	2	Injuries, not specified
	1	Explosives, specified
	1	Systemic diseases, specified
32. .2%	7	DISORDERS OF CONJUNCTIVA, specified
	2	Etiology, unknown to science
	1	Syphilis, prenatal
	1	Trachoma
	1	Trauma (including burns)
	1	Systemic diseases, specified
	1	Etiology not specified
33. .2%	6	MICROPHTHALMOS
	6	Congenital and hereditary, not specified
34. .2%	6	DEVELOPED AND DEGENERATIVE ANOMALIES, specified
	2	Congenital and hereditary, not specified
	2	Etiology undetermined by physician
	1	Septicemia
	1	Hereditary and familial
35. .2%	6	INTRAOCULAR HEMORRHAGE
	5	Etiology undetermined by physician
	1	Vascular diseases
36. .2%	5	CONJUNCTIVITIS
	3	Etiology undetermined by physician
	2	Infections, not specified
37. .1%	4	DEVELOPED AND DEGENERATIVE ANOMALIES, not specified
	2	Congenital and hereditary, not specified
	2	Etiology undetermined by physician
38. .1%	3	DISORDERS OF OPTIC NERVE, specified
	1	Infections, specified
	1	Hereditary and familial
	1	Congenital and hereditary, not specified
39. .1%	2	DISORDERS OF CONJUNCTIVA, not specified
	1	Trachoma
	1	Infections, not specified
40. .1%	2	ALBINISM
	1	Congenital and hereditary, not specified
	1	Hereditary and familial
41. .1%	2	REFRACTIVE ERRORS, specified
	1	Congenital and hereditary, not specified
	1	Etiology undetermined by physician
42. .1%	1	DEVELOPED ANOMALIES AND DEGENERATIVE CHANGES
	1	Syphilis, prenatal
43. .1%	1	ANOPHTHALMOS (excluding surgical)
	1	Etiology not specified
44. .1%	1	OBSTRUCTION OF CENTRAL ARTERY OR VEIN
	1	Vascular disease

CHART 2

ETIOLOGICAL INCIDENCE

569	Etiology undetermined by physician
406	Senility
321	Etiology unknown to science
226	Trachoma
160	Congenital and hereditary, not specified
87	Syphilis, not specified
65	Injuries, not specified
65	Vascular diseases
59	Infections, not specified
53	Infections, specified
51	Diabetes
45	Ophthalmia neonatorum, not specified
43	Hereditary and familial
42	Syphilis, prenatal
37	Nephritis and other kidney diseases
33	Trauma (including burns)
31	Nonindustrial injuries, specified
30	Meningitis
29	Toxic poison, specified
27	Toxic poison, not specified
26	Firearms
23	Smallpox
21	Measles
21	Explosives, specified
20	Neoplasms
19	Etiology not specified
17	Tuberculosis
16	Systemic diseases, specified
14	Systemic diseases, not specified
11	Scarlet fever
11	Injuries, incidental to surgery
10	Ophthalmia neonatorum, gonorrheal
9	Gonorrhea (excluding O.N.)
9	Syphilis, acquired
9	Typhoid
7	Explosives, not specified
7	Street and traffic accidents
6	Household activities
4	Anemia and other blood diseases
3	Birth injuries
3	Alcohol (methyl, wood, denatured, etc.)
2	Tobacco
2	Diseases of pregnancy and childbirth
1	Ophthalmia neonatorum
1	Septicemia
1	Play or sport
1	Industrial diseases (including poison)
1	Industrial injuries, not specified
1	Noninfectious diseases, central nervous system
1	Congenital and hereditary, prenatal
1	Consanguinity

NOTES, CASES, INSTRUMENTS

IODISM WITH SEVERE OCULAR INVOLVEMENT*

Report of a case

H. Krieger Goldberg, M.D.
Baltimore

The purpose of this report is to record a case of bilateral edema of the lids and conjunctivae, bullous keratitis, and hypopyon secondary to iodine poisoning. Such severe ocular involvement is rarely observed.

M. F., a white woman, aged 68 years, was first seen at the Johns Hopkins Hospital in February, 1905. She complained of stiffness of the hands and feet. For a year she had experienced tingling of the right foot and a pain which radiated to her right hip from the right leg. She had had fleeting pains in her arms. Also, she had noticed that she could not differentiate coins or buttons when placed in her left hand.

Complete examination, supervised by Dr. Osler, showed only astereognosis of the left hand and moderate ataxia of both arms. Attention was drawn to the peculiar personality of the patient by Dr. Osler, but no details were stated. During her stay in the hospital she was given a saturated solution of potassium iodide. The initial dose was 15 min. and this was increased 2 min. every other day. After four weeks the iodide was discontinued because an iodide rash appeared on the extremities and face. Four days after discontinuing the iodides, the symptoms had entirely disappeared.

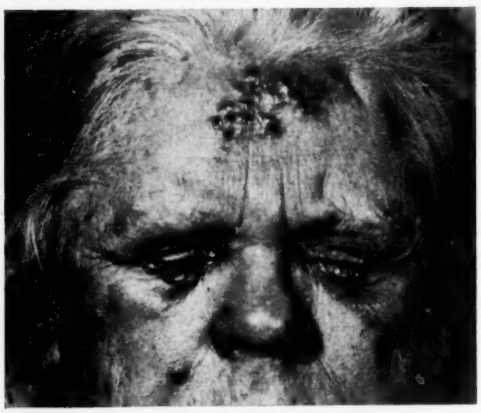
Following the patient's return to her home she continued to have "rheumatic" attacks and on her own initiative she took potassium iodide in the same dosage that had been given to her in the hospital. After using the drug for any length of time she invariably developed a skin eruption over the face and extremities. Sometimes the eruption was macular and at other times it was pustular or vesicular. Cessation of the drug always resulted in disappearance of the rash. The patient noted that with each succeeding skin eruption, a shorter period of iodine consumption was necessary to produce the rash. She had not used the iodides for about one year until in February, 1938, she commenced taking 30 min. t.i.d. with increasing doses every other day. After three weeks a vesicular skin eruption appeared over the face, arms (figs. 1 and 2), and legs. However, on this occasion the patient persisted in the use of the iodide. On March 8th her eyes became painful and her vision failed rapidly. She again entered the hospital on March 11th, when her vision was reduced to light perception in both eyes.

OCULAR EXAMINATION

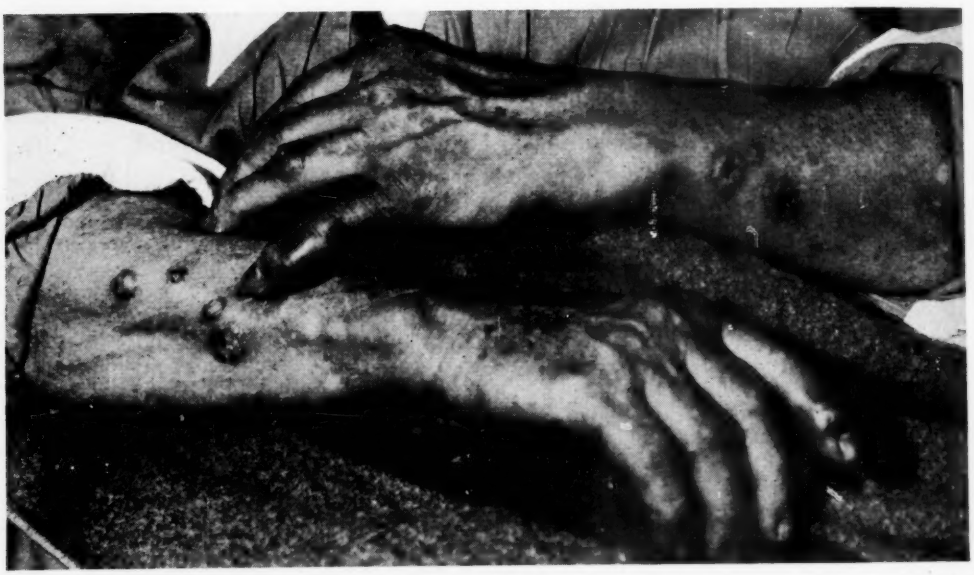
There was such edema and redness of the lids that they could not be opened voluntarily by the patient. The bulbar conjunctiva of the right eye was chemotic. The cornea was grayish and infiltrated. The iris was similarly discolored. The pupil was contracted, irregular, and adherent to the lens. There was a thin layer of exudate over the surface of the iris with loss of all normal luster and markings. There was a small hypopyon in the inferior angle of the anterior chamber. Tension was normal. The left eye presented a more advanced generalized chemosis. The cornea was grayish and hazy in color. Outlines of the pupil could barely be seen. There was a heavy

* From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University, Baltimore, Maryland.

exudate over the anterior surface of the iris and a large hypopyon half filled the anterior chamber. There was a definite loss of epithelium in a small area toward the 3-o'clock position which stained with fluorescein. Tension was normal. There



Figs. 1 and 2 (Goldberg). Cutaneous lesions of face and arms on admission.



was a bare suggestion of red reflex in both eyes.

Vision was light perception with good projection in each eye.

There were a few bullae over the right cornea and several very faintly staining areas where there had probably been other recent bullae. On the left cornea towards the center there were five or six small blebs which appeared to be on the posterior surface of the cornea and to arise between Descemet's membrane and the endothelium (fig. 3).

General physical examination was essentially negative except for the ocular and dermatological findings. The result of the neurological examination revealed the same ataxia of the arms and astereognosis of the left hand which had been found on her first admission. Her personality had not changed. She was inordinately proud of the fact that Sir William Osler had prescribed for her. This probably accounted for her continuing the prescriptions in spite of the repeated attacks of iodism. She definitely relished reciting the routine of her disability and her method of combating it.

The Wassermann reaction, blood gonococcal fixation test, blood chemistry, and blood cytology were entirely negative. Cultures taken from the bullae of the skin were sterile. The survey of various

systems was negative. Examination for iodine in the urine showed its presence in large quantities.

On March 11th, the day of admission, the patient was given instillations of atropine one percent t.i.d. and iodides were stopped. Immediately the patient made an unusual and rapid improvement. On March 12th the left cornea no longer stained. On March 15th the patient was able to count fingers at two feet with the right eye and the iris pattern was readily distinguished (fig. 4). On March 22d the vision was 20/200 in each eye. Nuclear sclerosis made it unlikely that further visual improvement could follow. The right eye was otherwise normal. The left eye at this time continued to show some folding of Descemets' membrane in the areas where the bullae had been observed. The skin eruption had entirely disappeared and iodine was no longer demonstrable in the urine. The patient continued to progress in an uncomplicated manner and therefore 14 days after admission she was discharged.

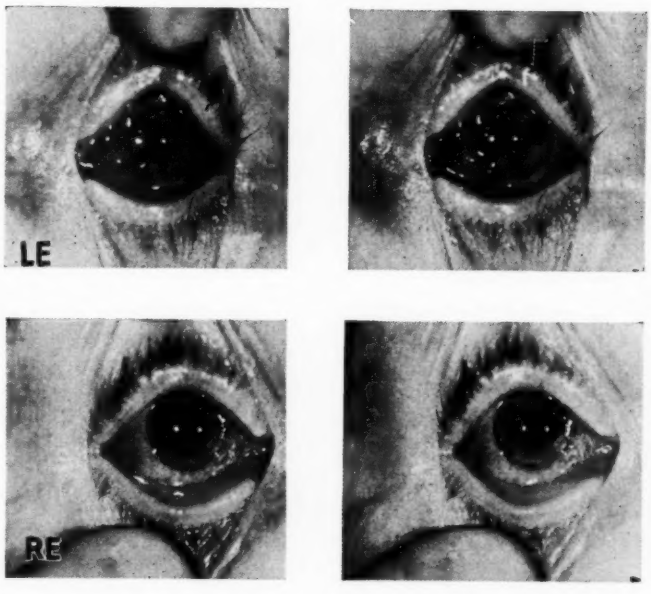


Fig. 3 (Goldberg). Stereoscopic photographs of right and left eyes on admission. Edema of conjunctiva, corneae, and bilateral hypopyon are present.

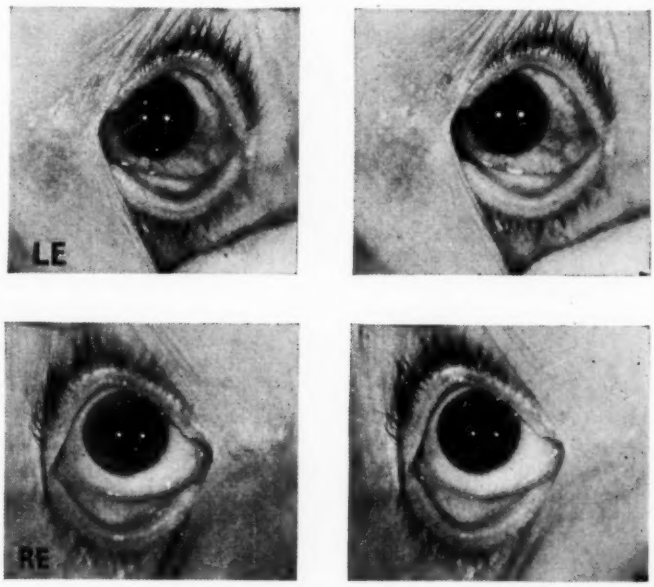


Fig. 4 (Goldberg). Stereoscopic photographs of right and left eyes nine days later.

Comment

Swelling of the lids and conjunctiva is frequently observed after iodides have been prescribed.[1] Such symptoms may appear without an iodide rash appearing. More severe ocular involvement has been observed. Lewin,[2] in 1899 and again in 1925, recorded retinal hemorrhages and corneal lesions resembling the bullae seen

on the skin as resulting from the use of iodides. Hallopeau[3] in 1899 described a case in which bullae of the cornea appeared, the cornea became almost entirely opaque and the pupils were fixed to light and were irregular. Discontinuance of the drug was followed by disappearance of the lesions which reappeared when the drug was again prescribed. The case here reported, although similar to that of Hallopeau, was even more severe. It would seem obvious that the ocular condition observed in the case here reported arose from lesions in the eye which were similar to those frequently observed in the skin. The hypopyon therefore resulted from the irritation of the corneal bullae which caused an irritative iritis and hence a sterile hypopyon.

Wilmer Ophthalmological Institute.

References

[1] d'Epenay, M., and Hallopeau, M. Ann. de derm. et syph., 1906, v. 7, p. 580.
[2] Lewin, L. Lehrbuch der Toxikologie. Verlag von Georg Stilke, Berlin, 1929, p. 106.
[3] Hallopeau, M. Ann. de derm. et syph., 1888, v. 2, p. 760.

SOCIETY PROCEEDINGS

Edited by Dr. H. Rommel Hildreth

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Section on Ophthalmology

February 11, 1938

Dr. Walter E. Camp, *president*

Report on the 15th International Congress of Ophthalmology at Cairo, Egypt

Dr. Henry P. Wagener, Rochester, Minnesota, said the recent International Congress of Ophthalmology differed from the meetings of most ophthalmological societies in that the major part of the scientific sessions were devoted not to discussions of ophthalmic surgery and therapeutics but to two phases of internal medicine which are closely linked to ophthalmology. The main subjects under discussion were hypertension and endocrinology, with especial reference to their ocular manifestations.

The ophthalmoscopic features and prognostic significance of the retinal lesions seen in cardiovascular renal diseases have been well established. The attention of the Congress was directed rather to the mechanism of development of these retinal changes. While Bailliart and Koyanagi incline still to the assumption of a direct action on the retina of a toxic product, presumably from the kidneys, most of the other speakers seemed to feel that the retinal circulation was involved primarily. It was recognized, however, that the lesions in the retina did not arise as a result of arterial or arteriolar sclerosis but through a dynamic disturbance of blood flow in the realm of pathologic physiology. There is still a lack of definite proof and agreement as to the exact nature of this disturbance of blood flow. Thus, Volhard adheres strongly to his theory of ischemia; Mylius, of Hamburg, believes that passive hyperemia on the venous side is the predominant lesion, and Fritz, of Brussels, introduces the idea of capillary anisoperfusion and proposes the name "anisospastic retinitis." Koyanagi presented excellent microphotographs of the lesions seen in "albuminuric retinitis," some of which showed marked lesions of the arterioles, which, however, he thinks, are due to invasion of the arteriolar walls by phagocytic pigment-epithelial cells. It is noteworthy, however, that the vascular lesions he demonstrated compare very closely with those found in the arterioles of muscles and of the kidneys.

Lobeck, of Jena, and Thiel, of Frankfort, reported on a series of measurements of the diameters of the retinal vessels in various types and phases of hypertensive disease. For these measurements, they used a new type of instrument which should be superior to the previous graticule methods and should give us a more accurate idea of what actually happens to the lumen of the vessels in hypertension.

A number of papers were presented on the use and value of measurements of the retinal arterial pressure with the dynamometer of Bailliart. This instrument seems to be more systematically used in Europe than in America and is said to yield significant results in investigations of suspected cerebral lesions and in lesions of the optic nerve and retina, whether associated or not with an elevation of systemic blood pressure. A paper that deserves further study along these lines was presented by Espildora-Luque, of Santi-

ago, Chile, on "Solitary retinal arterial hypertension."

The symposium on endocrinology brought out the fact that there is still much uncertainty as to the influence of specific endocrine insufficiencies on specific eye diseases. The difficulty inherent in endocrine studies was well brought out in the excellent summary of the various endocrine syndromes presented by Snapper, of Amsterdam. Professor Snapper emphasized the close interrelationship between the different endocrine glands and the difficulty of determining at times which of the glands is involved primarily. A paper of considerable interest was presented by Blatt, Bratianu, and their coworkers of Bucharest. They demonstrated roentgenographic changes in the sella in 28 of 37 cases of cataract and evidences of reduced pituitary function in all. Wibaut, of Amsterdam, reported good results from the treatment of retinitis pigmentosa with memformon (theelin).

Two days of the Congress were devoted to sessions of the International Organization against Trachoma and of the International Society against Blindness. The etiology of trachoma came up again for discussion. Thygeson, of New York, reported on his studies of the Halberstaedter-Prowazek elementary bodies which he believes to be the morphologic unit of trachoma virus. Apparently, trachoma is considered now to be most probably a virus rather than a bacterial disease. Busacca, of São Paulo, Brazil, and Cuénod and Nataf, of Tunis, reported the findings of infraorganisms of the rickettisia family in cases of trachoma. The campaign against trachoma and purulent ophthalmia in Egypt is apparently well organized, and very excellent work is being done by the Egyptian ophthalmologists in the treatment of these diseases in spite of the tremendous number of cases which must be handled.

Peculiar pupillary reaction to cold stimulation in Horner's syndrome

Dr. A. G. Athens, Duluth, reported the following case not only because of the unusual nature of its distinguishing symptom but also with the hope that, with careful study in the attempt to correlate the findings with our present knowledge of the underlying anatomy and physiology, it might be the means of throwing a little light on a somewhat confused subject.

A young medical student, aged 20 years, home on vacation, consulted him on December 20, 1937, because he had noticed for four weeks previously that, when he took a drink of cold water, the left pupil would dilate irregularly. On questioning he stated that the same thing occurred but to a less degree when he washed his face or took a bath in cold water. The right pupil was not so affected.

This patient had been treated by him in August and September, 1937, for a superficial marginal corneal ulcer of the left eye. This was treated with local applications, on two occasions, of 5-percent mercurochrome, and atropine, 0.5 percent combined with 1:6,000-mercury-bichloride ointment for a period of about three weeks. The pupil was back to normal size at the beginning of school on September 28th. There was no evidence of iritis, and the pupils were equal in size and reacted normally to light and convergence. The media were clear and the fundi negative. One month previous to the onset of the ulcer an abscessed tooth had been removed. Two years previously he had been treated for a similar corneal ulcer by another oculist. He was then under observation for one week. The oculist reports that he saw nothing otherwise abnormal about the eyes. Since that time the eye had been slightly injected at times.

At the age of 13 years, in 1930, he was taken to his family physician because of

obesity. His weight had been increasing abnormally for two or three years previously. He was somewhat listless, apathetic, and dull in school. Examination showed him to be 50 pounds overweight, to have a female distribution of the pubic hair, and underdeveloped genitalia. Basal metabolism was −9. A diagnosis of Froehlich's syndrome was made and the patient was treated with antuitrin and thyroid extract. After a few weeks there were definite signs of improvement, and the development thereafter appeared to progress along fairly normal lins.

On July 7, 1937, he was admitted to a hospital for study of a lymphadenopthy. It had been noted that various groups of lymph nodes had been enlarging over a period of five months. Examination revealed cervical, axillary, epitrochlear, and inguinal nodes greatly enlarged but not tender. Two nodes from the left cervical group were removed for examination. One of these proved to be caseous. Though a definite diagnosis of tuberculosis could not be made, this was strongly suspected, since the patient had had considerable contact with a maternal grandmother and two aunts who had had this disease. All serological and agglutination tests were negative. X-ray films of the chest were negative. Pulse and respiration were normal throughout his three weeks' stay in the hospital. X-ray therapy was used on the enlarged nodes.

Examination on December 20, 1937, revealed a rather tall, though normally developed, well-nourished young man, apparently a little worried about his condition. He was much interested in the examination and was very coöperative. He was of a rather emotional nature. Several moderately enlarged, fairly discrete, insensitive nodes were found in the neck below the mandibular angles on both sides. A distinct, firm mass measuring 3 by 2 by .5 cm. occupied the region of the left parotid gland. A similar but much smaller, bean-sized mass was felt in the corresponding position on the right side. These were freely movable under the skin and overlying structures, and were not tender. There was a small surgical scar one inch below the left ear.

The pupil of the right eye in ordinary light measured 4.5 mm. and reacted normally to light stimulus, convergence, and consensually. The upper lid drooped slightly, but the levator functioned normally. There was no enophthalmos. The pupil of the left eye was round and measured 2.5 mm. It contracted promptly and regularly to light and promptly returned to its original size. It also reacted promptly to convergence and consensually. In a semidarkened room it dilated regularly to 7 mm. while its fellow pupil dilated to 8 mm. A 4-percent solution of cocaine dilated the right pupil to 9 mm. and the left to 3.5 mm. Epinephrine hydrochloride 1:1,000 solution dropped into the left conjunctival sac, repeatedly, had no effect on the pupil. One drop of 1-percent homatropine hydrochloride dilated the left pupil and the greatest dilatation was medialward. About six weeks later a small drop of epinephrine injected beneath the conjunctiva of the left eye dilated the pupil quite widely.

When the patient took a swallow of cold water the pupil of the left eye dilated markedly, downward and inward, giving it a distinct pear shape. This followed immediately on taking the water and the pupil promptly returned to its former size and shape—the entire procedure occupying three or four seconds. The reaction resembled an ameboid movement. During the reaction the patient observed a "peculiar sensation" as though the vision dimmed slightly. Washing the face or dipping the hands into cold water produced the peculiar dilatation of the pupil but to a less degree. Warm water either

swallowed or on the hands or face did not affect the size or shape of the pupil. When pressure was made on the left anterior faucial pillar the reaction occurred but not so markedly as with cold water in the mouth. Considerable pressure over the cervical nodes and along the carotid sheath produced no effect on the pupil. Seven weeks after this phenomenon was first noted by the patient it suddenly could no longer be elicited.

The cornea of the left eye showed only a very faint marginal opacity at the site of the former ulcer. The corneal reflex was present. The iris showed no evidence of atrophy and was uniformily colored to match that of the right eye. The other media were clear throughout. The retinal vessels showed no increase in caliber in comparison with those of the right eye. Both fundi were otherwise normal. The tension was normal and no appreciable difference in accommodation between the two eyes could be determined. The visual fields were normal for form and colors. The extraocular muscles were in balance. Visual acuity measured 20/15 in each eye.

This patient presented an atypical picture of Horner's syndrome with the added bizarre pupillary dilatation on peripheral cold stimulation. The signs commonly described as characterizing Horner's syndrome are (1) miosis, (2) narrowing of the fissure from drooping of the upper lid, (3) enophthalmos, (4) unilateral absence of sweating, and (5) hemiatrophy of the face. To these physiologists have added (6) failure of the pupil to dilate with cocaine, (7) dilatation of the pupil with epinephrine in the conjunctival sac. This case differs, then, from the classical Horner's syndrome in the following details. Enophthalmos was not present. As this is by no means a constant or even a common finding in sympathetic paralysis it will not be further considered. Opportunity was not given at the first examination to study the effects of sweating. The pupil dilated slightly but definitely with cocaine. It did not dilate with epinephrine in the conjunctival sac. Peripheral cold stimulation caused, for a period of six weeks, a dilatation of the lower nasal portion of the pupil.

A fairly careful search of the literature has revealed few similar cases and no such reaction to cold stimulation. Carline observed transitory dilatation of one pupil which remained so for several hours or days. He concluded that the patient was hysterical. Wiesner reported the case of a woman, 60 years old, who had paralysis agitans and also typical Horner's syndrome, and whose pupils were "cat-like and dilated when she took a sharp breath." Erlenmeyer observed bizarre behavior of the pupils of a hysterical woman of 47 years. They continued to change from oval to round, dumb-bell shape, and even assumed ameboidlike movements. Coppez recorded the case of a girl who had mydriasis of one eye which changed to miosis when her head was bent forward. The miosis was accompanied by severe pain. The patient had mediastinal adenitis and Coppez believed that inclining the head caused the enlarged glands to press on the cervical sympathetic producing the pupillary change. Rosenfelt reported a case of carcinoma of the esophagus involving the cervical sympathetic trunk. Typical Horner's syndrome was present, and the pupil dilated markedly when pressure was made over the area. Ehlers reported a case of hippus associated with Horner's syndrome in acute anterior poliomyelitis. Cases of essential iris atrophy, with the pupil assuming, at different times, many shapes have been recorded by Harms, Casey Wood, deSchweinitz, Lane, Zentmayer, and McKeown. In all these cases the lesions were unilateral. While the pupil in this condition dilates irregularly, the ir-

regularity is due to contractures and distortions of the remaining live tissue. These cases, when followed, terminate in glaucoma. Schur studied 34 cases of cervical-sympathetic paralysis and found that psychic stimuli caused dilatation of the pupil on the affected side in all.

Any attempt to explain the atypical features in his case—namely, the behavior with cocaine, epinephrine, and cold stimulus—will naturally involve a review of the anatomy and physiology of the sympathetic control of the pupil. Unfortunately, this is not thoroughly understood and there is controversy on a number of points. Physiologists and neuro-surgeons have found that removal of the superior-cervical ganglion does not always abolish the sympathetics in the eye nor even those to the dilator muscle. They have found it necessary to remove, in addition, the superior-thoracic ganglion from which fibers may follow the vertebral artery, to end, according to Duke-Elder, in the ciliary ganglion, or to remove the ciliary ganglion itself.

DeTakats and Clifford, reporting the results of sympathectomy for retinitis pigmentosa in six patients, stated that in two of these, in which the operation consisted in removal of the superior-cervical ganglion, Horner's syndrome did not develop, and in one case an incomplete Horner's syndrome developed. The test used for complete denervation was failure of cocaine to dilate the contracted pupil. Schur found that cocaine caused a slight dilatation of the pupil in all his 34 cases. This, he thought, was due to relaxation of the sphincter. This view was first expressed by Kuroda in 1915 and differs from that of Duke-Elder, Adler, and others who accept the view that cocaine acts by exciting the sympathetic. Duke-Elder states that although the reaction to cocaine is present immediately after section of the postganglionic fibers it disappears after degeneration of the nerve.

The slight dilatation to cocaine, then, in his case can probably be accounted for on this basis and is not inconsistent with what can be expected in Horner's syndrome in an early stage. We should expect, however, this reaction to disappear soon.

We may now consider the failure of epinephrine to dilate the pupil in this patient. Epinephrine in 1:1,000 solution when dropped into the conjunctival sac does not dilate the pupil in the normal eye. When epinephrine is injected subconjunctivally or when the epinephrine content of the blood is suddenly increased the normal pupil will dilate. When the superior-cervical ganglion is removed the pupil is still more responsive to epinephrine, even when it is dropped into the conjunctival sac. Physiologists have long known that smooth muscle deprived of its nerve supply becomes hyperirritable to epinephrine. This appears to be a property peculiar to unstriped muscle supplied by sympathetic nerves. In such experiments on the eye the pupil is spoken of as "sensitized"—the "paradoxical pupil" of Langendorff. This sensitization appears to diminish with time although it is not completely abolished even after degeneration of the nerve fibers. Hartman and Loder and Itikawa found the effects of epinephrine upon the denervated pupil to be more striking when their experiments were performed within a few days after removal of the cervical ganglion. One would then expect epinephrine response, as cocaine response, to diminish after the nerve fibers had had time to degenerate. Unfortunately, in reports of clinical cases the time element, in regard to the effect of these stimuli, has been given very little consideration.

Arnold Knapp called attention to the effect of epinephrine on the glaucomatous

eye, where it dilates the pupil even before clinical signs of the disease have appeared. This has been accepted as a valuable diagnostic sign in early glaucoma and probably indicates a hyperirritability of the dilator mechanism.

Hubert recently reported several cases of Horner's syndrome to support his contention that it is necessary, in order to sensitize the pupil, that the lesion involve the superior-cervical ganglion or the postganglionic fibers. If the lesion, he contends, is in the cord or the preganglionic fibers the dilator muscle retains its connection with live nerve cells and will not be rendered hyperirritable to epinephrine. He advocates the use of epinephrine to localize lesions involving the sympathetic pathway. Byrne has shown, however, that lesions in the cord or brain stem also render the pupil hyperirritable to epinephrine, excitement, and so forth. Certain systemic diseases such as diabetes and exophthalmic goiter may render the pupil hyperirritable. A weak solution of cocaine previously instilled into the eye appears to sensitize the pupil to epinephrine. This may, at times, account for the erroneous impression that epinephrine dilates the normal pupil.

It is difficult to correlate in this case the reaction to cocaine and the absence of a reaction to epinephrine. It is suggested, as a speculation, that cocaine was more completely absorbed from the conjunctiva than epinephrine and that, had the patient been seen earlier, when the muscle was perhaps more irritable, a reaction to epinephrine would have occurred.

Why did the pupil dilate only on one side? It is possible that, due to a difference in development or a slight atrophy on the temporal side, this portion lost its irritability sooner than that which reacted. Another possible explanation may be found in the peculiar enervation of the dilator muscle. Braunstein showed that by stimulating the temporal long ciliary nerve, dilatation of the outer upper half of the pupil is produced, and after severing the temporal nerve and stimulating the cervical sympathetic, dilatation of the inner lower half of the pupil occurs. It is possible to believe that the fibers of these two nerves were so separated in the cervical chain or plexuses that those composing the nasal nerve were the ones that escaped, for a time, the destructive lesion. Indeed there is considerable experimental evidence that the pupillodilator and vasomotor fibers run from the ciliospinal center in separate paths.

The patient was reëxamined on February 11, 1938. There was a definite hemiatrophy of the left side of the face, and this side remained dry while the right side perspired. There appeared to be a definite enophthalmos even after the drooping lid was lifted. However, none was found with the exophthalmometer, and the appearance of enophthalmos could doubtless be attributed to hemiatrophy of the face. Cocaine no longer dilated the pupil, neither did the pupil dilate spontaneously in the dark. It was therefore reasoned that the reaction of the sensitized pupil to cocaine and in the dark is of a transitory nature.

Discussion. Dr. Henry P. Wagener said studies related to the sympathetic nerve supply to the eye are always of considerable interest. The exact origin of these nerves and their mode of action have not been fully determined as yet. As Dr. Athens has pointed out, a complete Horner's syndrome is not produced in most cases by a simple removal of the cervical-sympathetic ganglia. If the anterior roots of the first and second thoracic ganglia are sectioned also, ptosis, miosis, and failure of the pupil to dilate with cocaine will result in most cases. Enophthalmos is not, however, an early effect of such an operation. If enophthal-

mos occurs at all, it would seem to be the late result of atrophy of the orbital tissues associated with the facial hemiatrophy.

The effect upon the general sympathetic system of local application of cold is also of considerable interest. It has been shown by Hines and Brown that immersion of the hand in ice water for one minute will cause an elevation of blood pressure that is greater in the hypertensive or prehypertensive individual than in the normal person. Hines and Brown thought that a widespread vasopressor reaction occurred through the mechanism of a neurogenic reflex arc. That the reaction was not due to increased secretion of adrenalin was shown by its occurrence in adrenalectomized dogs and in patients with Addison's disease. The reaction occurs also in patients around whose arm a tourniquet is placed to shut off the return flow of blood from the hand in the ice water.

The dilatation of the pupil which occurred in Dr. Athens's patient following the local application of cold at a distant point presents further evidence of a widespread stimulation of the sympathetic nervous system by cold applied to a relatively small area of the periphery. The dilatation of the pupil was not necessarily a response to an increased secretion of adrenalin.

Etiologic factors in myopia

Dr. T. R. Fritsche, New Ulm, Minnesota, stated that the causes of axial myopia have been the subject of considerable discussion and many varied and diverse explanations have been given. Various authors have advocated such theories as acquired disease of the posterior segment, uric-acid diathesis, gravity theory, endocrine imbalance, calcium deficiency, epinephrine deficiency, and focal infections as being causes of myopia. Some have denied the influence of heredity, some believe it to be due to lues or tuberculosis in the parents, some believe that measles plays an important role, some stress the importance of acquired lues, while others believe that it only augments an already existing myopia. The mechanical theory has many advocates, who believe that the oblique muscles and the rectus muscles in their action compress the globe and cause it to bulge posteriorly at the same time that the optic nerve exerts a pull on the posterior globe during convergence. There are those who believe that myopia may be due to a tendency to undue expansion or delay in the toughening process of the sclerotic. Others believe that it may be due to excessive near work or strain, if either eye has had some slight injury. The theory has been advanced that a deficiency of adrenocortical substance may result in an increased formation of aqueous humor which will lead to glaucoma if the eyeball is strong, or to myopia if the sclerotic coat is weak. The observation has been made that it does not occur among the savage races and that it is most prevalent in those who do prolonged close work during the growth period.

Statistics taken by Schleich on 300 infants eyes showed all to be hyperopic. A summary of the examination of 2,400 eyes of small children in Germany showed only nine to have myopia. Stocker in Lucerne examined 4,614 eyes of children between the ages of 7 and 16 years, without cycloplegics, and found 8.4 percent to be myopic. Callan found an incidence of 1.2 percent myopia in Negro children in grade schools and an incidence of 3.4 percent in high schools, out of a total of 457 children who were examined. A statistical study in the United States showed no myopia under 5 years of age, an incidence of 8.1 percent between the ages of 5 and 10, and an incidence of 25.7 percent between the ages of 10 and 20.

Children's eyes are normally hyperopic

and this hyperopia decreases as maturity is reached, due to the growth of the eyeball. It is only natural that there are normal variations in the size of the eyeball, some of which may even be large enough to result in moderate degrees of myopia; yet a myopia of this type should not be considered pathological or due to any of the numerous causes given above. Likewise there are many cases in which the larger size of the eyeball may be purely hereditary and as such the myopia cannot be considered as being due to any of the various causes. Those eyes should be considered as pathologically myopic, in which the myopia appears or progresses at a rate faster than the normal growth rate of the eye.

In the following series of cases the majority had a considerable degree of myopia ranging from 1 to 10 diopters; two cases are included in which there was mixed astigmatism and in which the minus meridian exceeded the plus meridian. During the year 1934 there were 35 cases of congenital lues on record at Ancker Hospital. Several of them were in infants and consequently were not tested. Twenty-eight patients, in age between 3 and 20 years, were refracted under cycloplegia. Sixteen of the 28 (or 57 percent) were found to be myopic, the other 12 (or 43 percent) were found to be normal or hyperopic. Eight of the 35 (or 23 percent) of these patients with congenital lues had interstitial keratitis, and all eight in this particular group had a considerable degree of myopia. In one half of these cases of congential lues with myopia there were somewhat similar errors of refraction in the two eyes. The others had a considerable difference, both in the degree of myopia as well as a decided difference in the axis of the astigmatism.

It is hard to say what influence treatment of the congenital lues has on the course or incidence of the myopia. All of the cases at Ancker Hospital were under observation or treatment. There was a record of only one patient, aged 19 years, who had had a refraction test two years before, and his myopia was found to be increased by one-fourth and one-half diopter, respectively, in right and left eyes. Whether there would be a greater incidence of myopia among undiscovered and consequently untreated cases of congenital lues cannot be determined from this series of cases. Likewise it would be interesting to refract these patients again and observe their progress.

Two interesting cases were seen that were not in the Ancker series. These were a sister, aged 29 years, and a brother, aged 24 years. Both father and mother were hyperopic, and both were luetic at the time of the birth of the girl, who developed interstitial keratitis and malignant myopia, with the left eye much worse than the right. The boy, who was born after the parents had been under some treatment, did not develop interstitial keratitis. He has two diopters of simple myopic astigmatism at axis 180 degrees in each eye.

Because of the fact that there was a definitely increased incidence of myopia among congenital luetic patients, it was thought worth while to look for evidences of congential lues among children and young adults afflicted with myopia. In addition to looking for the usual signs of congenital lues, such as Hutchinson teeth, chronic middle-ear deafness, interstitial keratitis, rhagades, saber shins, and fissures on palms and soles, peculiar formations of the cranium, and a routine Wassermann test, the eyes were inspected with a slitlamp for loss of pigment on the pupillary border, and luetin tests were made. The luetin test is a skin-sensitivity test for the Spirochaeta pallida that has

been discarded for many years in favor of the Wassermann and its modifications. The diagnosis of congenital lues in the absence of positive serology, definite clinical signs, or knowledge of active lues in the parents is practically impossible, and on this account it is thought that the luetin reaction might prove to be valuable. After examining 25 myopic patients and finding nothing more than a few having decreased or absent pigment of the pupillary margin and two questionably positive luetin reactions, he became discouraged and discontinued doing all the extra work on the routine refraction cases. These three were aged 29, 37, and 50 years. All three had negative Wassermann reactions and positive luetin tests. The parents of the two older cases were dead and there was nothing in the history to suggest lues. The younger patient had bilateral chronic otitis media. His mother was dead, but his father was living; a Wassermann test was negative, and the father likewise denied any luetic infection. Viewed with the slitlamp all three of these persons had interstitial keratitis typical of that seen in the interstitial keratitis of congenital luetics.

Just how the congenital syphilis acts to produce an increased incidence of myopia is a debatable question. Possibly there may be a low-grade luetic scleritis. At least in the cases of interstitial keratitis, in which one actually sees the disease of the cornea and which often results in the stretching or distortion of the cornea, one could readily imagine that this disease could exist in the sclera as well as in the cornea and result in an enlarged eyeball. Likewise a person afflicted with congenital lues can be assumed to be constitutionally inferior and naturally would be more apt to suffer some endocrine or nutritional disturbances which may act in some way to weaken the sclerotic coat of the eyeball. In determining the size of a given eyeball, two factors must be considered. In the first place there is a hereditary factor. This factor determines a tendency toward a certain size of eyeball, as well as a tendency toward a certain degree of toughness of the sclerotic coat. The second factor is the intraocular pressure. Both the intraocular pressure as well as the toughness or tensile strength of the sclerotic coat may be altered by numerous conditions. The intraocular pressure may be altered by endocrine, vasomotor disease, physical or mechanical conditions. The toughness of the sclerotic coat may be altered by growth, disease, trauma, endocrine, and nutritional causes. Thus in the final analysis it can be seen that pathological myopia is not due to any one cause, but to a number of conditions working together, and that a condition which is most important in producing one case of myopia may be the least important in another.

George E. McGeary,
Secretary

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

February 28, 1938

DR. CLIFFORD WALKER, *president*

SOME PRACTICAL CONSIDERATIONS IN THE INTERPRETATION OF VISUAL FIELDS

DR. HAROLD F. WHALMAN opened his discussion with the admonition to observe standard conditions when taking visual fields, emphasizing particularly the necessity for adequate and constant illumination. For this he preferred artificial light, which, of course, can easily be controlled. He pointed out the importance of the quantitative method of plotting the perimetric fields and indicated that his prefer-

ence for this purpose was the tangent screen and a series of test objects ranging from 1 mm. to 40 mm. in size. The patient should sit at a distance of two meters from the point of fixation.

Dr. Whalman then presented various characteristic field defects. He pointed out the difference between the neurasthenic and the hysteric fields. In neurasthenia the field is a spiral, starting out at approximately normal limits in the periphery and gradually decreasing in extent as each meridian is tested until the field has come in close to the point of fixation. In hysteria the concentrically contracted field for one test object shows no increase in extent when larger test objects are used.

In retrobulbar neuritis green may be lost entirely while the field for red is reduced considerably. The papillomacular bundle is frequently involved, giving a central or paracentral scotoma. Toxic amblyopia may be considered a chronic variety of retrobulbar neuritis with the involvement beginning in the ganglionic retinal cells and then spreading to the nerve fibers of the papillomacular bundle. This again produces paracentral scotoma. Bizarre fields are produced by chorioretinitis. In primary optic atrophy there is an early and rapid loss of the red and green fields as well as the nasal form fields.

Dr. Whalman then outlined the possibilities of field changes in hypophyseal tumor, stating first that in some of these cases there is an early occurrence of paracentral scotoma due to a toxic effect on the papillomacular bundle. Later the effects of pressure or stretching of the chiasmal crossed fibers can be detected by the appearance of quadrant anopsia and hemianopsia. A field which starts as a bitemporal defect might later be changed into a homonymous defect by the bursting through of a pituitary stroma to one side of its confines, producing pressure on only one optic tract. It is possible also for unilateral blindness to occur with a normal or partially changed field on the other side.

Discussion. Dr. Clifford Walker stated that 50 percent of pituitary tumors produce homonymous field defects rather than bitemporal, so that the latter cannot be considered typical of pituitary lesions without cognizance of the former.

A TREATMENT FOR CHRONIC DACRYOCYSTITIS

DR. PAUL SOUTHGATE read a paper on this subject which was published in this Journal (October, 1938).

Harold F. Whalman,
Editor.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

February, 1938

DR. ALEXANDER G. FEWELL, *chairman*

THE ASSOCIATION OF AN ANNULAR PIGMENT BAND ON THE POSTERIOR CAPSULE OF THE LENS WITH A KRUKENBURG SPINDLE

DR. WILLIAM ZENTMAYER reported the case of a boy aged 16 years, who showed in each eye a characteristic Krukenberg spindle. On the surface of the posterior capsule of the lens of each eye there was an annular band of brown pigment. As seen with a +20 D. lens of the ophthalmoscope it had an average width of .5 mm. and was situated about 2 mm. in from the equator of the lens. The margins of the band were more or less serrated. In the right eye the circular band was broken at points corresponding to the 8- and 4-o'clock positions, the gaps being about 2 mm. wide. At these breaks short tags of pigment were seen directed toward the tips of the ciliary processes.

In the left eye the band was unbroken, but there were a few pigment lines running toward the equator of the lens. The refraction was a compound myopic astigmatism.

Cases from literature were cited in which Krukenberg spindles were seen to develop in eyes that were without inflammatory changes.

The anomaly on the posterior capsule of the lens is explained by Mann as being due to the maintenance of the contact between the tips of the ciliary processes and the lens for too long a period. The ciliary ring increases in diameter more rapidly than does the lens, with the result that the ciliary processes are withdrawn from the lens and a space is left between the two.

In view of the cases of Krukenberg spindle cited and the opinion expressed by several other writers as to the pathogenesis of this lesion, the author concludes that in the case reported it seems reasonable to believe that granules freed from the pigment line on the posterior capsule of the lens were carried by the circulation of the fluids of the eye into the anterior chamber, where the thermal currents deposited them on the posterior surface of the cornea.

Discussion. Dr. Alfred Cowan said that Krukenberg spindle is of much more frequency than it was considered to be before the advent of the corneal microscope. Very often the same type of pigmentation that is found in Krukenberg spindle can be seen with the slitlamp, but it would escape notice by the ordinary methods of examination with oblique illumination. The pigment is deposited all over the posterior surface of the cornea and the spindle formation is the result, merely, of a denser arrangement over the spindle-shaped area. While it may be that the pigment-ring imprint on the posterior surface of the lens is congenital in most instances, he has seen this condition as the result of inflammation several times.

Delayed removal of magnetic foreign bodies from the vitreous

Dr. Charles R. Heed detailed the history of three patients and emphasized the importance of accurate localization and early extraction of magnetic foreign bodies, before the advent of degenerative changes which, he believes, result in an overwhelming percentage of all cases where an ultraconservative policy has been pursued. He advocates a scleral incision when the metal is located behind the lens, and counsels against the introduction of any magnet tips into the vitreous.

Case D. B. was examined five days after the accident. Vision O.D. was 20/100. There was an incomplete coloboma at the 11-o'clock position, due to an operation by the company physician to remove particles of torn iris, on the day following the accident. A small wound of the cornea, iris, and lens periphery was found with a posterior star opacity. A magnet extraction was made through the scleral incision on March 11, 1937. Two months later the vision was 20/20, and the eye was quiet. Ophthalmoscopic and slitlamp examinations exhibited a less dense opacity of the posterior subcapsular star.

Case B. R. was examined June 23, 1937, 112 days after the accident. The pupil was dilated; the iris was copper colored, and pigment cells were seen on the anterior nasal quadrant posterior to equator. Diagnosis: retinochoroiditis with symptoms of siderosis. After localization, a metallic body, 1.5 mm. in size, was extracted by means of a magnet through the scleral incision. At the last visit, July 20, 1937, vision was 20/30. No deposits on the capsule were seen, and only a few vitreous opacities.

Case T. J. A. was examined six days after the injury. The patient was myopic, with corrected vision of O.D. 20/20, O.S. 20/200. A particle of metal from an iron pin or hammer had entered the left eye through the upper lid. The X ray located the metal in the vitreous. Vision became blurred in 48 hours and there was severe pain three days later. A delicate haze in the vitreous and a somewhat blurred disc were seen, but there was no apparent retinochoroidal lesion nor hemorrhage. The slitlamp showed marked wrinkling and folds of Descemet's membrane, many cells in the aqueous, no apparent change in the lens, and an increase of cells in the vitreous. Diagnosis: iridocyclitis. Localization demonstrated a metallic foreign body, spherical, 2 mm. in diameter, in close proximity to the ora serrata, 4 mm. to the nasal side of the median line. A magnet extraction through the scleral incision, on the nasal side of the superior rectus, about 8 mm. posterior to the limbus, was made. There was no prolapse of vitreous or pigment. Severe pain was experienced for two hours. The patient slept well and was free from pain on the following morning. He was discharged on November 8, 1937; when examination showed vision in the left eye, corrected, to be 20/50. By means of the slitlamp, a few fine vitreous opacities were observed, but no hemorrhage nor visible retinochoroidal lesions. Descemet's membrane was clear and free from wrinkles, the capsule and lens were clear, and there were no cells in the aqueous. A report from his physician made two months later, gave vision, corrected, as 20/20.

Discussion. Dr. H. Maxwell Langdon reported on a patient, whom he had seen about twelve years ago. She had been to one of the large hospital clinics after having had a needle strike the eye from a machine on which she had been sewing. She herself suggested an X-ray examination, but was told there was no foreign body in her eye. Not satisfied with this, she went to a private physician, who also assured her that an X-ray examination was unnecessary. By this time, her employer had notified the insurance company. The X ray showed a part of the needle, over one-half inch long. It had been in the eye almost a week when he saw her. Fortunately, a magnet attracted the end of the needle and it came out through the posterior scleral wound readily. The result was an eye with a vision of 5/9.

Another case which had always interested him was that of an employee of the Pennsylvania Railroad in its shops at Olean, New York. This man felt something strike his eye while at work and very promptly an X-ray film was taken, which was said to be negative. The eye was free from inflammation, the vision was normal, and he was told that there was no foreign body in his eye. In a short time he returned saying that he had no pain and could see; but he was sure there was something in his eye and the same procedure was gone through again with negative findings. He returned again after an interval of time but was sent to Philadelphia. Dr. Newcomet localized a very small foreign body, not as large as the head of a pin, which was easily removed with a magnet, and the patient returned to work, there never having been any inflammation of the eye and the vision never having been less than 6/6.

Unless the foreign body is in the anterior chamber, Dr. Langdon favors removal by the posterior route.

Dr. Leighton F. Appleman agreed with Dr. Heed that foreign bodies should be removed as soon as possible after their presence within the eye has been established. It is not always possible to see a foreign body within the eye, due to hemorrhage; therefore X-ray examination should always be made. The amount

of intraocular disturbance may vary considerably, depending upon the position of the wound of entrance and the size and shape of the penetrating particle. Also the subsequent reaction will depend upon whether infection has been introduced with the foreign body. Clean, magnetic bodies can often be removed through the wound of entrance; if this is not possible, an incision through the sclera may allow of its passage after the tip of the magnet is presented at the opening.

Dr. Walter I. Lillie said that he had removed an intraocular foreign body situated in the vitreous of the right eye of a young man. It had entered the vitreous through the lower nasal sclera, just posterior to the ciliary body. His convalescence was uneventful, and the eye is now normal, except for a small area of healed choroiditis at the site of entrance and exit of the foreign body. His vision of 3/60 can be improved only to 6/60 with a +.50 D. sph. ⊂> +1.00 D. cyl. ax. 90°. The visual-field examination was quite interesting, inasmuch as he would only see the 10-mm. test object in the extreme opposite field from any avenue of approach. This, of course, does not conform with any type of organic field change, or that associated with amblyopia ex anopsia, or hysteria. Dr. Lillie is sure that the patient is malingering, and such a situation should always be thought of in a compensation case.

THE LATE RESULTS FROM CERVICAL SYMPATHETIC RESECTION IN RETINITIS PIGMENTOSA

DR. E. B. SPAETH reviewed the results of seven cases and said that while no definite statement can or could be made, there are some indications present which seem to suggest that this form of therapy may be seriously considered in certain cases.

Discussion. Dr. William Zentmayer said that if the pathology of pigmentary degeneration of the retina is what we believe it to be and the disease is an abiotrophy, he would not expect more than a transient improvement in the vision from a sympathectomy.

WARREN S. REESE,
Clerk.

SAINT LOUIS OPHTHALMIC SOCIETY

February 25, 1938

DR. ROY MASON, *president*

METASTATIC ACTINOMYCOTIC CHOROIDITIS

DR. JULE ELZ read a paper on this subject which will be published in this Journal.

Discussion. Dr. Harvey D. Lamb said that Dr. Elz is very fortunate in getting such an unusual specimen. For that matter, any eye with a metastatic or endogenous inflammation, that can be examined anatomically in the florid stage, is valuable. Dr. Elz's case with this organism is the only one of its kind ever to have been reported. Axenfeld in 1894 stated that in cases of metastatic septic endophthalmitis, if the involvement was bilateral, the primary change was usually in the retina. If only one eye was affected, the choroid, as a rule, was the first coat of the eye to be inflamed. Axenfeld, however, could give no explanation why in one individual the retina and in another the choroid should be the primary seat of the septic process in the eye. The septic emboli in the retina are very readily discharged from the retinal blood vessels into the vitreous. From the vitreous, the irritant coming in contact with the ciliary body and retina causes the formation of pus cells from these coats. As a consequence, the vitreous receives many pus cells and the eye is hopelessly lost. Dr. Elz demonstrated that in his case there was a primary deposit of the organism

in the choroid. He early noted clinically a complete detachment of the retina. The pus cells are all subretinal because they come from the infected choroid. In addition the choroid is generally densely infiltrated with pus cells. A panophthalmitis can occur from septic involvement of the retina or from a septic choroiditis.

Indirect choroidal tears

Dr. Leslie C. Drewes read a paper on this subject which will be published in this Journal.

Discussion. Dr. Harvey D. Lamb said that Dr. Drewes has shown how important it is, in a rupture of the choroid, to take a field of vision. Certainly, in one case that he saw very early, with hemorrhages that covered the retinal blood vessels, there must have occurred a tearing of the retinal tissues. It is novel to have a rupture of the choroid, without a tearing of the lamina vitrea, as Hagedoorn found anatomically. If there exists a simple separation of the pigmented epithelium of the retina, it is no choroidal rupture. The main trouble is that we do not have enough anatomical examinations of eyes with so-called choroidal ruptures.

Dr. B. Y. Alvis said the point was made that the yellow-pink portion of this tear resembled the lesion of the retina. That being the case it would seem that it should have destroyed the maculopapular bundle and have cut down the central vision very materially. The central part of the field should have been lost. It is more likely to be a lesion of the superficial portion of the choroid; whereas the white portion involves the entire choroid and the retina.

Dr. Drewes said the fact that this optic-nerve atrophy was noticeable in a week after the injury indicates the damage of the nerve fibers. Quite a number of such cases have been reported in the literature and about 20 cases of incomplete nasal scleral tear.

Nonoperative improvement of divergent strabismus

Dr. Vincent L. Jones stated that an improvement in exotropia without operation is not unusual. The two cases here presented, being of 40 and 50 degrees divergence, are considered sufficiently unusual to warrant reporting them.

The first, a boy, 19 years of age, had a 40-degree divergence. Vision in the right eye was 20/24 and with —0.75 D. cyl. ax. 180° equalled 20/16. Vision in the left eye was 20/32, and with —0.50 D. cyl. ax. 180° equalled 20/16. A recession of the external rectus of the right eye was performed in October, 1936, and orthoptic exercises were instituted shortly thereafter.

In February, 1937, the patient's eyes were straight when fixating near objects, but the right eye diverged slightly when fixating distant objects. A second operation was advised, which the patient refused. However, orthoptic exercises were continued until the present, and now he is wearing comfortably a moderate overcorrection for the compound nearsighted astigmatism. His eyes are straight when fixating distant and near objects, no suppression of either eye is observed, and the fusion is good.

The second case is that of a 35-year-old woman who had an alternating divergent squint for years. When first examined the divergence was 50 degrees, and there was no fusion. Vision in each eye was 20/30, and with —0.50 D. sphere equalled 20/20. Operation was advised but she refused, so an effort to determine if any improvement could be accomplished by exercises was made and continued to date with more or less regularity. Her eyes are now straight, and her fusion is good; she can read comfortably with four prism diopters, base out, before each eye.

These two cases, and particularly the second one, demonstrate very clearly

what a surprising improvement can be obtained in many cases by rather simple orthoptic exercises.

Discussion. Dr. M. L. Greene said he was interested in these cases because he very often saw such conditions when examining patients for the service. There was one case of a young man who had a definite tendency to divergence at times. He could not be measured for adduction, for as soon as prisms were in place, the eyes would diverge. He was overcorrected and in three or four months took another examination and passed the eye test.

Dr. B. Y. Alvis said that following Dr. Greene's suggestion in a paper presented here some time ago, he tried overcorrection on a child 12 or 13 years old who had a divergence with a tendency to diplopia and who was not able to do at all well in school. The overcorrection was about 1.50 diopters with a good result, and the patient developed a very satisfactory degree of stereopsis. The thing that is puzzling in these periodic divergent cases is that a person may have a perfectly good stereopsis and still at times get an external deviation without diplopia, while at other times the eyes will be parallel and show good muscle function.

H. Rommel Hildreth,
Editor.

AMERICAN JOURNAL OF OPHTHALMOLOGY

The New Year witnesses the Journal's embarkation on its twenty-second year. Due to the loyal support of the profession it is now possible to publish a bigger and better journal than ever before. The increase of paid subscriptions by more than 200 in the past two years has permitted the continuance of the larger issues introduced in January, 1937. If one may judge from the material now on hand awaiting publication the coming volume promises to be one of the best. The editors take this opportunity to thank the many contributors of original articles, and the abstractors who have devoted so much time for the benefit of others to reading, in many cases translating, and digesting the literature, to present it in condensed form for our readers. It is frequently hard work and always time consuming. To them go the most sincere thanks of the editor and for them he wishes an especially happy New Year.

This year as heretofore an effort will be continued to present material of practical value to the ophthalmologist so that in each issue there will be much that the clinician can use in his daily work.

Letters were sent out in December to nonsubscribers, suggesting that the Journal would make a welcome gift to a friend or an ideal present for a family to give the ophthalmologist husband or father. This is a thought that we would like to convey to our subscribers, too. If you can help enlarge the subscription list you will do a service to the entire profession as well as to yourself, for new subscriptions permit the publication of a better magazine.

The past year, which marked the initiation of the third decade of the publica-

tion of the combined ophthalmic journals now known as the American Journal of Ophthalmology, furnished an occasion for a very enjoyable luncheon of the editors collaborators, and stockholders, at the meeting of the American Medical Association in San Francisco. A similar subscriber's luncheon in Saint Louis this spring is being planned. It is hoped that there will be a large attendance.

Nineteen thirty-eight may not have been the best of years for business and profession, and we may not like the socialistic trend in medicine, but as we look the world over we can find a great deal for which to be grateful. So here's for a prosperous and happy New Year.

Lawrence T. Post.

SCIENCE AGAINST DICTATORSHIP

Scientific discoveries of facts or general laws are made by independent thinkers who test new ideas by their knowledge and experience and then submit them to the judgment of others best qualified to appreciate them. The progress of the world has depended on the free thought of great men. Shall it now be stopped to increase or sustain the power of dictators? Science, the growing knowledge of truth, now meets the challenge of dictatorship. Helpful guidance may be needed by children, but a time comes when each must choose, direct, and live his own life. Military power and political authority must not limit nor dictate to science.

Science has now gained a position in human service and direction equal to the influence formally exerted by custom, tradition, or statute law. Scientific truth is more powerful than any ruler. From the days of Vesalius and Harvey medical science has advanced more than under 1500 years of the dictatorship of Galen. The advance of science comes by the great thoughts of free men. Such thinkers break down the traditions of the past and are foes of tyrrany. Dictators cannot tolerate them.

Ophthalmology has contributed to, and shared in, the advances of modern science. Can it continue to do so under dictatorship that burns all books written by Jews and exiles all whose thinking it cannot control? If Hitler had then ruled Germany would Helmholtz, a direct descendant of the pacifist William Penn, have been appointed to teach physiology in the Naval Medical School at Kiel and been permitted to invent the ophthalmoscope? If communists had continued to control Paris, would the devout Catholic, Louis Pasteur, have been allowed to save the silk industry of France, exterminate anthrax, and prevent rabies?

Freedom of independent thought, and to teach what is proved, are more important to scientists than grants of money for research, or laboratories, or professorships, or titles of nobility. Germany and Italy have shared in the development of modern ophthalmology. But they cannot continue to do so when they refuse to hear teachers utter new scientific truths. Already the literature that comes in German or Italian journals is less important than it was before teaching positions were placed under Nazi and Fascist control. France, Britain, and America now take the leadership in scientific investigation and study of ophthalmology. Holland, Belgium, Switzerland, Scandinavia, Finland, and, perhaps, the Soviet Union, must be looked to for really important contributions to future ophthalmic literature.

Edward Jackson.

AMERICAN BOARD OF OPHTHALMOLOGY CHANGE IN METHOD OF EXAMINATION

With steady increase in the number of

candidates presenting themselves for examination, one of the most important problems in the work of the American Board of Ophthalmology is as to the type of examination which will most satisfactorily determine whether or not the candidate is entitled to the Board's certificate.

The Board has kept before it consistently the principle that its investigation of professional qualifications must seek to discover whether the candidate may safely be entrusted with the care of important ocular disorders, including errors of refraction and those serious conditions which call for surgical intervention.

In the earlier years of the Board, it was found practicable to divide the examination into two parts, both occurring on the same day. The first part was an oral examination which was completed by lunchtime. The second part was a written examination, held in the afternoon.

With much larger numbers of candidates to deal with, the Board decided a few years ago to omit the written examination, and to depend (so far as examination was concerned) entirely upon oral tests. While much has been said in criticism of any form of examination as a criterion of educational qualifications and ability, examinations do furnish information which it is difficult to obtain by any other means. Certain defects are inherent in oral examinations, other defects may be attributed to a written examination. To some extent each form of examination is complementary to the other.

After careful deliberation, the Board has now decided to use a new plan for examination of candidates. A practical and oral examination will be held, as before, at the time of meeting of the American Academy of Ophthalmology and Otolaryngology and of the American Medical Association, and also occasionally in relation to other important medical assemblies.

Sixty days prior to the date of the practical and oral examination, written examinations will be held simultaneously in various cities throughout the United States. The subjects covered in the written examination will include all the subjects previously covered by the Board's oral examination. It is intended that the first written examination shall be held on Wednesday, March 15, 1939, and candidates will of course receive due notice as to the places of examination.

Each candidate must take the written examination and must pass it satisfactorily before he may appear for the practical and oral examination. (He must also have presented acceptable case reports before appearing for the final examination.)

The oral examination to be held in Saint Louis on May 15, 1939 (at the time of the meeting of the American Medical Association in that city), will deal only with the following six practical subjects: External diseases, Ophthalmoscopy, Pathology, Refraction, Motility, and Practical surgery.

The Board announces that all applications for the written examination which is to be held on March fifteenth must be on file at the Board's office, 6830 Waterman Avenue, Saint Louis, not later than February 15, 1939. All correspondence on the subject must be addressed to that office.

Candidates whose applications are already in the hands of the Board will receive direct notification in regard to the new arrangements.

W. H. Crisp.

BOOK NOTICES

THE PRINCIPLES AND PRACTICE OF PERIMETRY. By Luther C. Peter, M.D. Fourth edition, cloth-bound, 332 pages, 222 engravings, and 5 colored plates. Philadelphia, Lea & Febiger, 1938. Price $4.50.

This fourth edition of probably the best known book on perimetry by an American author is rather similar to the preceding editions. It is therefore apparently published because of the exhaustion of previous editions rather than for the presentation of new material in perimetry that has been brought out in the last few years. However, even small additions are valuable, as it is always of advantage to keep textbooks strictly up to date.

The subject of perimetry is obviously of very great importance. It is surprising how few of the candidates for the American Board of Ophthalmology are well prepared in this subject, although undoubtedly they do better now than formerly. For some reason they have seemed to think that this subject is very simple and that a vague idea of general principles is all that is necessary.

Peter's book presents the subject logically and clearly. It is very readable and covers the field adequately. The first chapters are devoted to anatomy, physiology, and technique; the central division to general pathology of the visual field; and the latter part to the special pathology of the fields and various functional nervous diseases. The illustrations are good, being clear and easily understandable. It is a valuable book for the student of eye diseases and for the practicing ophthalmologist.

Lawrence T. Post.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is only mentioned in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision.
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

7

UVEAL TRACT, SYMPATHETIC DISEASE, AND AQUEOUS HUMOR

Gallino, Juan. **Rubeosis iridica.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 311-313.

The author reports a case of rubeosis of the iris in one eye of a diabetic patient 65 years of age. Some cases reported in the literature are mentioned. The author believes the condition to be produced by proliferation of blood vessels in the course of a torpid iritis.

Ramon Castroviejo.

Meyer, G. P. **Uveal tuberculosis.** Jour. Med. Soc. New Jersey, 1938, v. 35, March, p. 138.

Meyer discusses the diagnosis and treatment of uveal tuberculosis and states that specific desensitization is the most important curative measure.

Theodore M. Shapira.

Miklos, Andor. **The late prognosis of operated cases of iridocyclitis.** Graefe's Arch., 1938, v. 139, pt. 2, pp. 403-412.

Of twenty eyes with chronic iridocyclitis, repeated paracentesis or therapeutic iridectomy failed to improve and even made conditions worse in thirteen eyes. Six of the eyes with favorable results had an iridocyclitis of rheumatic origin. Among six eyes with chronic iridocyclitis, cataract extraction was performed in five eyes, with failure by the extracapsular method and success intracapsularly in only one eye, in which the cyclitis had been stationary for years. Beside these cases, the author's material included forty eyes on which similar operations had been performed with analogous results. Iridectomy should therefore be performed in tuberculous iridocyclitis only when a secondary glaucoma cannot be relieved by conservative measures.

H. D. Lamb.

Mohamed, I. A. **Studies of a series of cases of sympathetic ophthalmia.** Bull. Ophth., Soc. Egypt, 1936, v. 29, p. 168.

A study of 27 cases of sympathetic ophthalmia is presented. Eight of the cases followed perforating or rupturing injuries, and ten occurred postoperatively, nine following iridectomy and one after cataract extraction. Nine cases were not related to trauma. The minimum time of development was eight

days and the maximum time four years. The histopathologic findings are summarized. Edna M. Reynolds.

Scardaccione, Mario. **Urotropin in the therapy of sympathetic ophthalmia.** Boll. d'Ocul., 1938, v. 17, Feb., pp. 112-123.

Ten cases are reported. Six occurred after penetrating wounds of the eyeball and four after cataract operations. The age of the patients varied from 5 to 73 years, and the onset of sympathetic symptoms from twenty days to three months after the accident or operation. Urotropin was given by daily intravenous injection of 10 c.c. of a 40-percent solution. Better effects were obtained the sooner the treatment was started. In advanced and grave cases the effect was nil even in cases in which urotropin was used with other therapy. In four favorable cases the vision obtained was from 5/10 to 10/10, while in other cases the symptoms subsided with preservation of the sympathizing eye. (Bibliography.) Melchiore Lombardo.

Van Lint. **Sympathetic ophthalmia evolving during fourteen years.** Bull. Soc. Belge d'Opht., 1938, no. 76, p. 25.

The left eye was injured in 1924 and was enucleated thirteen days later, at which time the right eye had begun to show signs of cyclitis. This yielded to treatment but recurred in 1927, 1934, and again in 1936. Vision was reduced to one-third and treatment failed to improve it. One member in discussing the paper expressed the belief that the case might be one of tuberculous iridocyclitis lighted up by trauma.

J. B. Thomas.

Weekers, L., and Reginster, H. **Contribution to the study of recurrent hypopyon iritis.** Bull. Soc. Belge d'Opht., 1938, no. 76, p. 31.

Dermatologists have recently called attention to a relapsing syndrome peculiar to women and characterized by thrush, acute ulceration of the vulva, and iritis. In some cases reported in men ulcers of the scrotum occurred in association with thrush and iritis. The authors report at length the case histories of two patients, one male and one female. The etiology is uncertain, but the evidence seems to indicate an infectious, allergic lesion. Blood transfusion has proved especially useful. It is noted that the ocular lesions include uveitis and not a simple iritis or even iridocyclitis. J. B. Thomas.

Yanes, T. R., and Ferrer, O. **Spontaneous bilateral uveitis with dysacousia, alopecia, and poliosis.** Rev. Cubana de Oto-Neuro-Oft., 1938, v. 7, Jan.-Feb., p. 5.

The authors review in some detail the literature on the subject of uveitis associated with dysacousia, alopecia, and poliosis, and report two additional cases with complete clinical and laboratory findings. Edward P. Burch.

8

GLAUCOMA AND OCULAR TENSION

Allmaras, Fritz. **Observations on a glaucoma family.** Zeit. f. Augenh., 1938, v. 95, Aug., p. 276.

The author gives a family tree in which eleven members of two generations had chronic simple glaucoma. Eight of the patients were women. He adds very brief notes on the eyes of the affected members. The four who were operated upon reacted well to trephining. This glaucoma form seems to be inherited directly and dominantly.

F. Herbert Haessler.

Barkan, Otto. **Glaucoma: classification, causes, and surgical control.** Amer.

Jour. Ophth., 1938, v. 21, Oct., pp. 1099-1114.

Bordeaux. **The surgical treatment of glaucoma.** Bull. Soc. Franç. d'Opht., 1937, v. 50, p. 142.

The author seeks amelioration and cure of glaucoma in its various forms by means of a modified Elliot technique. Under local anesthesia, a conjunctival flap is dissected down to the limbus, and the episcleral fascia is incised. With the galvanocautery, an opening is then made in the sclera, the point of contact of the cautery being 1 mm. from the insertion of the conjunctiva. By successive applications the hole is enlarged to 3 mm. The charred tissue is curetted away, a thin spatula is introduced, and the root of the iris and the angle of Schlemm are freed. The flap is replaced and sutured.

The author used the method sixty times, in all types of glaucoma. In 47 patients the tension was restored to a value of 25 mm. Hg (Schiötz) or less. In several cases a repetition of the operation was necessary to obtain the desired result. Clarence W. Rainey.

Fahmy, A. Y. **Histopathology of a case of capsular glaucoma.** Bull. Ophth. Soc. Egypt, 1936, v. 29, p. 164.

A case of glaucoma capsularis of Vogt in a patient of 75 years is described. There were capsular exfoliations of the lens, with fluffy felt-like tufts at the pupillary margin. No increase in tension was observed even under dilatation. Microscopic sections showed refractile granular masses on the posterior surface of the iris, with pigment granules and refractile masses adherent to the thickened anterior lens capsule. Edna M. Reynolds.

Fialho, Abreu, Jr. **Spontaneous oscillations of ocular tension.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 315-324.

The author studied normal and glaucomatous eyes to determine the behavior of ocular tension throughout the day. The Schiötz tonometer was used. The nine patients presented normal eyes, and eyes with chronic simple, absolute, or secondary glaucoma. Fifteen conclusions are presented, the most important being as follows: Ocular tension varies during the day in both normal and glaucomatous eyes. As a rule, tension is higher in the morning than at night. Variations of tension are greater in glaucomatous than in normal eyes. In about 20 percent of normal eyes these variations do not occur. In only 8 percent of the eyes with chronic simple glaucoma were there no variations of tension during the day.

Ramon Castroviejo.

Mueller, Friedrich. **As to insistence upon treating glaucoma without operation.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 337-341.

The author discusses the different medical treatments reported in the literature for the care of glaucoma. The effect of irritation of the sympathetic upon ocular tension is also briefly discussed. Ramon Castroviejo.

Mueller, Friedrich. **Glaucoma and pseudoglaucoma.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 333-336.

The author discusses briefly some reports of Salzer and Thiel concerning glaucomatous symptoms in eyes without increase of ocular tension. According to the author, the term "pseudoglaucoma" or "glaucoma without increased tension" is incorrect. These are

really eyes in which instead of true glaucoma, there are other changes which simulate glaucomatous changes in the visual fields. Such field changes may be due to pressure upon the optic nerve by extraocular vessels or to degeneration of the optic nerve on account of circulatory disturbances.

Ramon Castroviejo.

Nemeth, Lajos. **The constitution of glaucoma patients.** Klin. M. f. Augenh., 1938, v. 101, Aug., p. 222.

Fifty male patients with primary glaucoma from the eye clinics of Berlin and Budapest were followed up for several years with regard to types of constitution. Inflammatory glaucoma predominated in pyknotics, simplex in the asthenic type. In asthenics glaucoma sets in earlier, can be less influenced, and progresses more rapidly than in pyknotics. The blood pressure in asthenic glaucoma patients averages lower. Great climatic fluctuations render the therapy of a glaucomatous attack more difficult. In asthenics quieting of the nervous system, in pyknotics treatment of the vascular system is essential for preventing an acute attack.

C. Zimmermann.

Poos, F. **Uveal vascular reactions and intraocular pressure.** Klin. M. f. Augenh., 1938, v. 101, Aug., p. 210.

Under conditions simulating inflammatory hypotony, the author attempted experimentally to increase and decrease locally the hydrostatic pressure of the blood in the capillaries. He also observed the behavior of intraocular tension. His results justify the assumption that the fluctuations of intraocular tension after toxic damage to the uveal terminal circulatory channels, and at first also in every acute inflammation, are due to fluctuations of hydrostatic pressure in the capillaries.

C. Zimmermann.

9

CRYSTALLINE LENS

Berens, C., and Bogart, D. **Immediate operative complications of cataract.** Rev. Cubana de Oto-Neuro-Oft., 1938, v. 7, Jan.-Feb., p. 27.

A brief analysis with respect to the immediate operative complications of 1,004 cataract extractions performed at the New York Eye and Ear Infirmary is presented, with recommendations as to their prevention.

Edward P. Burch.

Borley, W. E., and Tainter, M. L. **Influence of dinitrophenol on the production of experimental cataracts by lactose.** Amer. Jour. Ophth., 1938, v. 21, Oct. pp. 1091-1098.

Bourne, M. C., Campbell, D. A., and Pyke, M. **Cataract associated with an hereditary retinal lesion in rats.** Brit. Jour. Ophth., 1938, v. 22, Oct., pp. 608-613. (See Section 10, Retina and vitreous.)

Bueckler, M. **Concerning the pathogenesis of zonular cataract.** Klin. Woch., 1938, v. 17, Sept. 17, p. 1325.

Two different groups must be distinguished. The pathogenesis of one is endogenous, of the other exogenous. An endogenous origin must be assumed for those zonular opacities which have their seat in the embryonic nucleus and are often hereditary. An exogenous genesis in connection with a constitutional pathologic condition must be assumed for the zonular cataracts, always bilateral, which are located in more or less extensive cortical zones around the embryonic nucleus and which therefore were developed post partum.

The etiologic factors which produce this latter group are largely unknown. The author could not confirm a regular association of this type of cataract with rickets or infantile tetany, two factors to which general opinion attributes it. For solution of this problem more exact classification and sharp separation of the various types are necessary.

Bertha A. Klien.

Bunge, E. **The cholesterin content of normal and cataractous human lenses.** Graefe's Arch., 1938, v. 139, pt. 1, pp. 50-61.

Forty-four normal and fifteen intracapsularly extracted cataractous lenses were investigated as to their content of cholesterin. During life, the total amount of cholesterin in the normal human lens increases to about five times that in the new-born. Lenses with mature cataract show a normal absolute amount of cholesterin.

H. D. Lamb.

El-Atawi, M. A. **Ectopia lentis.** Bull. Ophth. Soc. Egypt, 1936, v. 29, p. 159.

A case of ectopia lentis is described in which the right lens was dislocated up and out and the left lens down and in. A brief discussion of the frequency of ectopia lentis, the position of displacement, its causes, clinical signs, and treatment is included.

Edna M. Reynolds.

Fahmy, A. Y. **Elschnig-Török-Stanculeanu's intracapsular extraction.** Bull. Ophth. Soc. Egypt, 1936, v. 29, p. 136.

The results of 160 cases of intracapsular cataract extraction are reported.

Edna M. Reynolds.

Finlay, C. E. **Treatment of incipient cataract by means of vitamin C.** Rev. Cubana de Oto-Neuro-Oft., 1938, v. 7, March-April, p. 33.

The author reviews experimental work on the relationship between cataract and avitaminosis with respect to vitamin C, and tabulates his own results in treatment of cataract with vitamin C. Careful determinations were made of the concentration of the vitamin in the blood and urine, the methods of determination being described. The cases treated are divided into two groups, those with vision of 20/100 or better and those with vision less than 20/100. Of the former group of nine patients, six exhibited definite and three doubtful improvement. Of the latter group none were benefited.

The author concludes that in senile cataract there is a deficit of vitamin C and glutathione in the lens which increases as the lens opacity progresses; that this deficit may be demonstrated in the blood and urine; that, if the cataract is not advanced, benefit may be expected from ingestion of some source of vitamin C either orally, intravenously or by conjunctival instillation; and finally that there is a little hope of success from this method of therapy in advanced cases. Edward P. Burch.

Hoffmann-Rötzel, F. W. **Erythropsia of aphakics.** Zeit. f. Augenh., 1938, v. 95, Sept., p. 323.

The aged author, who had both eyes operated upon for cataract when he was sixty years old, has had ample opportunity to observe erythropsia. He describes in detail his observations of color phenomena. He feels sure that erythropsia is not a phenomenon of excessive illumination as claimed by some, but depends on an achromatism which results from dispersion of light rays. The cause is purely physical and depends on the presence of an excess

of red rays in the light of the rising and setting sun and that reflected from the snow. F. Herbert Haessler.

Laval, Joseph. **Bilateral congenital ectopia lentis with arachnodactyly (Marfan's syndrome).** Arch. of Ophth., 1938, v. 20, Sept., pp. 371-374.

After a brief résumé of the various signs and symptoms of this syndrome, two cases with this condition are reported. The author discusses the terminology and suggests that the term dysmesdactyly be adopted when the chromosomes concerned with mesodermal tissue are involved and dysmesectopia when the ectodermal tissue is also affected. J. Hewitt Judd.

Purtscher, Ernst. **Clouds of crystals in the clear senile lens-nucleus.** Graefe's Arch., 1938, v. 139, pt. 2, pp. 358-366.

Among seven patients, between 65 and 80 years of age, there were observed in the otherwise clear sclerotic lens-nucleus collections of very fine crystals, needle- or plate-shaped. In the beam of the slitlamp, they varied from red to green in color. The position of the crystals in the lens nucleus differed, but they were generally more numerous in the vicinity of the lens axis near the surface of the nucleus.

H. D. Lamb.

Rosner, L., Farmer, C. J., and Bellows, J. **Biochemistry of the lens. 12. Studies on glutathione in the crystalline lens.** Arch. of Ophth., 1938, v. 20, Sept., pp. 417-426.

Evidence is presented indicating that the difference in potential between the cortical and nuclear portions of the lens, found by potentiometric studies, is due to a change in the concentration of glutathione. Studies of the concentration of glutathione in the lens indicate that the concentration of the nucleus remains relatively constant while that of the cortex varies significantly with age. When animals were fed galactose, the glutathione in the lenses diminished. In young animals the appearance of cataract followed shortly after the loss of glutathione, while in older animals such a loss might occur long before the appearance of opacities. The sharp change in potential found with small variations in the glutathione-beta crystalline ratio may be a more sensitive method than actual chemical determination for estimation of glutathione when it is present in small quantities. J. Hewitt Judd.

Zeiss, Erich. **Lens changes from the action of ultrasound waves on extracted cattle lenses.** Graefe's Arch., 1938, v. 139, pt. 2, pp. 301-324.

Ultrasound waves being those in the air of more than 35,000 alternations per second are not transmitted to the human tympanic membrane. Ultrasound waves in this experiment are produced by causing a disc of quartz to vibrate by subjecting it to a high frequency current of electricity. The extracted cattle lenses placed in Ringer's solution and subjected to the ultrasound waves for from two to twelve minutes present vesicle-like opacities deep in the cortex. Longer ultrasound waves produce coarser or larger vesicles in the lens. When normal vitreous humor from cattle is subjected to these waves, it is found to liquify. H. D. Lamb.

10

RETINA AND VITREOUS

Allen, T. D. **Detachment of the retina.** Arch. of Ophth., 1938, v. 20, Aug., pp. 307-314.

This review includes a summary of

the theories on etiology and of the types of treatment devised to correct these conditions by the surgeons having the greatest experience, especially Gonin, Lindner, and Arruga.

J. Hewitt Judd.

Bourne, M. C., Campbell, D. A., and Pyke, M. **Cataract associated with an hereditary retinal lesion in rats.** Brit. Jour. Ophth., 1938, v. 22, Oct., pp. 608-613.

Attention was attracted, some three years previous to the compilation of this article, to rats affected with such cataract, the rats being in a research laboratory for breeding purposes and not for experimental procedure. The rats appeared otherwise well, and reproduced as expected. A hereditary factor was sought. A cataractous female rat with a litter of eleven was first received for study, and six generations of rats were bred and studied. The cataract appeared in successive generations. There were no deaths other than from middle-ear disease or "rat pneumonia." Diet and housing were all that should be expected for normal rat living. The progress of the cataract is described, age limitations for the various stages being noted. A definite cause has not been found. It is not due to disturbance of blood supply or from metabolic interference. The point that some abnormality in the vitreous body or in the lens capsule may be more directly to blame for the cataract than the retinal lesion itself is being more fully investigated. (See next abstract.) (References, figures.) D. F. Harbridge.

Bourne, M. C., Campbell, D. A., and Tansley, K. **Hereditary degeneration of the rat retina.** Brit. Jour. Ophth., 1938, v. 22, Oct., pp. 613-623.

(See preceding abstract.) There being few records of hereditary retinal defects in animals, the authors regard as of considerable importance this opportunity of studying the degeneration of such retinae through all stages. The first obvious appearance of the abnormality was at 21 days of age, while the terminal stage was noted at eighteen months or older. The degeneration is fully described through the various stages. A comparison of this hitherto unknown pathologic condition in rats with human retinitis pigmentosa is made, with the explanation that the resemblance is too close to pass unnoticed and unmentioned. (Figures, references.) D. F. Harbridge.

Danielson, R. W., and Long, J. C. **A case of retinal arterial occlusion with partial recovery.** Amer. Jour. Ophth., 1938, v. 21, Nov., pp. 1264-1265; also Trans. Western Ophth. Soc., 1937, 4th mtg.

Dubois-Poulson. **Oscillometry of the arteries of the extremities in thrombosis of the central retinal vein.** Bull. Soc. Franç. d'Opht., 1937, v. 50, pp. 224-233.

The authors classify their cases into three groups, according to the oscillometric index. When the index was low, the presence of hypertension of the artery indicated diminution in caliber of the vessels, due to sclerosis. When the index was low, and the arterial pressure was normal, the subjects were young persons, who often complained of cold sensations, pallor of the extremities, and increased sweating. The author thinks that juvenile arteritis may account for these cases.

When the index is elevated in all four extremities, it is difficult to decide whether the trouble is vascular, or is caused by disturbed heart action. When the index is raised in the lower vessels

alone, its peripheral origin is undeniable. The index is elevated when there is vascular hypotony. It is augmented by hypertension.

Clarence W. Rainey.

Conciliis, Nicola de. **Retinal metabolism in narcosis.** Boll. d'Ocul., 1938, v. 19, Jan., pp. 45-51.

The writer tested the effect of a narcotic of the uretan series on the retina of the ox. In different test tubes he put constant quantities of retina and methylen blue and phosphate in different concentrations. A solution of 1 to 5000 ethyl uretan was then added, while a physiologic solution was added in control tubes. The narcotic effect of the ethyl uretan was evidenced by the fact that five times as long was required for the retina to discolor the methylen. The results of the experiments are given in tabulated form, showing that the retina is highly sensitive to the narcotic action of the ethyl uretan. This is probably due to the fact that the retina is rich in lipoids. Melchiore Lombardo.

François, Jules. **Circinate degeneration of the retina and thrombosis of the macular veins.** Bull. Soc. Belge d'Opht., 1938, no. 76, p. 54.

The author reports two cases in each of which the circinate degeneration of the retina followed thrombosis of the superior macular vein. He calls attention to the fact that a macular vein and not a more important branch of the central vein of the retina was thrombosed. The circinate degeneration began three or four months after the initial thrombosis, and on the opposite side. (2 illustrations, 21 references.)

J. B. Thomas.

Fritz. **The blood volume in the retinal vessels.** Bull Soc. Franç. d'Opht., 1937, v. 50, p. 209.

The blood volume in the retinal vessels appears as integration of all the factors affecting retinal circulation. The ease with which one can cause a granular current in the retinal vein is a measure of blood volume in the retinal artery. Physiologically, when one compresses the globe more and more, one sees in the retinal vein at a certain point a rapid current which is interrupted completely when the retinal artery collapses. After a certain pressure, Mn, corresponding to the index of suppleness of the artery, diastolic collapse of the artery is increasingly noticeable, whereas the amplitude of systolic pulsation decreases, to be effaced entirely at a pressure Mx. This progressive increase in pressure reduces the passage of blood in an arithmetical progression. It is as if the pressure were constant, with the cross section of the artery normal at Mn, and zero at pressure Mx.

If the volume of blood entering the retinal artery is large, the venous column is not fragmented until the pressure is near Mx, but if the volume of blood in the retinal artery is reduced from any cause, then fragmentation occurs in the vein at some pressure near Mn. The author uses as a criterion, the speed of venous globules of one disc diameter per second. He expresses the blood volume by the formula

$$D = k + k'\left(\frac{C.V.G.-Mn}{Mx-Mn}\right)$$

in which k and k′ are constants, k being the volume when the fragmentation appears spontaneously in the neighborhood of Mn, and k+k′ being the volume when fragmentation occurs at Mx.

The normal value of blood volume in the retinal artery occurs when venous collapse coincides with the second half of arterial collapse, and averages 0.7. When fragmentation occurs in the first

half of arterial compression, a pathological condition is present.

Clarence W. Rainey.

Fritz. **Functional examination of the circulation of blood in the retinal vessels.** Bull. Soc. Belge d'Opht., 1938, no. 76, p. 45.

The author comments in detail upon the factors involved in an examination of the retinal circulation, such as degrees of pressure, differential pressure, caliber of the retinal artery, blood pressure in the retinal capillaries, and collapse of the artery. (2 tables, 25 references.) J. B. Thomas.

Gandolfi, C. **Bilateral blindness from anatomical and functional alterations in retinal circulation.** Rassegna Ital. d'Ottal., 1938, v. 7, May-June, pp. 287-300.

The author reports two cases of bilateral acute blindness occurring in elderly individuals, and which fall into the classification of occlusion of the central retinal artery. He discusses the affections of the retinal arteries from the point of view of etiopathogenesis and distinguishes two groups, functional and anatomical. The ophthalmodynamometer of Bailliart was used and the value of this instrument in such cases is stressed. Basing his data on clinical findings, and especially the study of pressure in retinal artery and vein, he shows that one case was functional and the other anatomical, that is, due to a lesion of the vessel wall.

Eugene M. Blake.

Genet, L., and Charpentier, R. **Retinal spasm in fatal diseases, from lack of acetylcholine.** Bull. Soc. Franç. d'Opht., 1937, v. 50, pp. 215-223.

The authors report treating with acetylcholine a 34-year-old man who had a fatal angina pectoris. Their observations of the retinal arteries did not enable them to differentiate the grave forms of the disease. The views of physiologists are reviewed. The term "chemical transmitters" designates those substances which permit transmission of nerve impulses from a neuron to a muscular or glandular cell or to another neuron. The point of action of these substances is the point of junction. Two such substances are known. One is adrenalin, which acts on the sympathetic nerves; the other is acetylcholin, which acts on parasympathetic nerves. When acetylcholin is lacking, vascular spasm occurs. Acetylcholin was supplied in the hope of relaxing vascular spasm.

Clarence W. Rainey.

Gibson, G. G. **Clinical significance of the retinal changes in leukemia.** Arch. of Ophth., 1938, v. 20, Sept., pp. 364-370.

This report is based on the study of 22 cases in which the clinical diagnosis of leukemia was confirmed in nine cases at autopsy and in seven additional cases by biopsy. The ophthalmologic diagnosis and prognosis of leukemia are discussed and attention is called to the close parallelism between the amount of hemorrhage in the retina and the degree of anemia which is associated with the leukemia. The evidence presented suggests that therapeutic and investigative procedures should be directed toward the anemia instead of the leukocytosis in the unsolved problem of leukemia. (Discussion.)

J. Hewitt Judd.

Klien, B. A. **Retinitis proliferans.** Arch. of Ophth., 1938, v. 20, Sept., pp. 427-436.

Several cases are presented to show that on the basis of clinical and histo-

logic studies it is possible to distinguish two types of retinitis proliferans and to deduce from the location and appearance of the plastic lesions the nature of the primary disturbance. In the first type, the exudation of hemorrhage from the retina into the vitreous, caused by inflammatory or traumatic alterations of the retinal vessels, is a primary event, and organization of the extravasation leads to the formation of the strand and membranes. In the second type, the main factor in the production of the plastic lesions is a slow circulatory impairment due to degenerative vascular disease, with formation of new compensating anastomotic channels, with or without hemorrhages at first. The retinitis is produced mainly by disturbance of venous circulation due to sclerotic changes of neighboring structures and preëxisting unfavorable topographic anatomic relationship. The differential diagnosis between the two types is clearly outlined and illustrated by fundus drawings and photomicrographs. J. Hewitt Judd.

Kurz, Otto. **Clinical aspect and pathogenesis of nonmyopic detachments of the retina (fixed detachment, retinal cysts, retinal splitting).** Graefe's Arch., 1938, v. 139, pt. 2, pp. 326-357.

In fixed detachment of the retina, the retinal tear is not so important, the retina is often transparent and the detachment vesicle-like or flat. Where ruptures exist, they frequently occur as separations at the ora serrata. Cysts in the retina are the primary factor, resulting either from traumatic influence or from congenital defects of development. Twenty-two such cases are tabulated in two groups. There are further tabulated seven cases (one bilateral) in which vesicle-like detachment of the retina was associated with peripheral choroiditis and was therefore apparently caused by inflammatory exudate. Among 27 cases of retinal detachment operated upon by the method of Weve (diathermy-puncture), 21 were healed.

H. D. Lamb.

Kurz, Otto. **Eye changes in lupus erythematosus.** Zeit. f. Augenh., 1938, v. 95, Sept., p. 315.

In a 24-year-old woman with lupus erythematosus, eye complications appeared during the three or four weeks before death. A phlyctenule-like papule developed at the limbus in the left eye, and four or five white protruding spots overlying the blood vessels appeared in the foveal region of the left fundus, accompanied by minimal edema and an occasional minute hemorrhage. Two days later, similar lesions appeared in the right fundus. In sections from the eyes obtained eight hours post mortem, it was seen that the retinal lesions consisted of gangliform degeneration of the nerve fibers. These clinical and histologic findings in a patient suffering from a severe septic affection characterize simple retinitis septica. It may occur as a transient lesion and end in complete recovery.

F. Herbert Haessler.

Lijo Pavia, J. **Vitreous humor, incarceration in one eye and deposits on hyaloid membrane of each eye.** Revista Oto-Neuro-Oft., 1938, v. 13, Jan., p. 15.

The author describes a cone-shaped vitreous condensation occurring in one eye and whitish deposits on the hyaloid membrane of both eyes, in 31-year-old syphilitic patient. Edward P. Burch.

Paton, R. T. **Recurrent retinal and vitreous hemorrhages in the young—Eales' disease.** Arch. of Ophth., 1938, v. 20, Aug., pp. 276-285.

The clinical manifestations of this

condition are reviewed and two cases are presented showing quite different fundus pictures. Both recovered, one with normal vision in each eye, and the other with restoration of two thirds of his sight in spite of a partially vascularized vitreous. The fundus changes are shown by drawings.

J. Hewitt Judd.

Perera, C. A. **Retinitis pigmentosa with "hole" in the macula.** Arch. of Ophth., 1938, v. 20, Sept., pp. 471-474.

The literature is reviewed and this combination is reported in a 16-year-old girl whose parents were first cousins. The macular lesion developed in one eye while the patient was under observation. The author suggests that both the peripheral and central degenerative lesions were the results of local vascular disease and retinal deterioration.

J. Hewitt Judd.

Trantas, Nico. **Operation for retinal detachment by diathermy coagulation, after the method of Weve. Retinal tears without detachment.** Bull. Soc. Franç. d'Opht., 1937, v. 50, pp. 234-239.

Success of operation for retinal detachment with a tear depends upon the best possible measurement of the location of the tear, and closure of the tear. The author takes the position of the ora serrata as basis for measurement. This is obtained by transillumination of the globe, placing the cone of the Lange lamp upon the opposite end of the lid, and conducting the ophthalmoscopic examination through a pupil dilated with atropin. Scuffing of the cornea by use of the contact glass is avoided by turning down over the cornea the conjunctival flap which is made in order to lay bare the sclera over the operative site. Incisions of the sclera at the site of the needle punctures aid in puncturing the softened eyeball. Among sixteen cases operated upon there were twelve cures, one was improved, and three not benefited.

The author reviews reports of five cases of tear without detachment, and adds case reports of five more such cases studied by himself.

Clarence W. Rainey.

Valois, Jeandelize, Drouet, and Lemoine. **Recurrent retinal hemorrhage, and endocrine disturbances.** Bull Soc. Franç. d'Opht., 1937, v. 50, pp. 267-280.

A nineteen-year-old woman, soon after the birth of her first child, had recurring hemorrhage in each eye, together with cessation of the menses. As long as administration of thyroid was kept up, the hemorrhages improved and failed to recur. Addition of hypophyseal substance to the treatment caused recurrence of the menses.

Clarence W. Rainey.

Vogt, A., Wagner, H., and Schneiter, M. **Investigations on the orientation of the normal structural membranes of the vitreous.** Klin. M. f. Augenh., 1938, v. 100, Aug., p. 235.

From thirty observations on normal youthful persons, the authors conclude that in erect position of the head the orientation of the structural membranes of the anterior vitreous is determined by the force of gravity.

C. Zimmermann.

Wibo and Rans. **Prepapillary floating opacity of the vitreous.** Bull. Soc. Belge d'Opht., 1938, no. 76, p. 11.

The authors report a case of prepapillary floating opacity of the vitreous observed in a girl nine years old having atrophy of both papillae and discrete

areas of choroiditis. This condition is said to occur exclusively in adults.

J. B. Thomas.

11

OPTIC NERVE AND TOXIC AMBLYOPIAS

Benedict, W. L. **Optic neuritis and retrobulbar neuritis: etiology and treatment.** Jour. Michigan State Med. Soc., 1937, v. 36, Dec., p. 946.

Benedict reports 500 cases of definitely proved multiple sclerosis, in 15 percent of which there was complaint of visual loss as the first symptom. In 40 percent visual disturbances were mentioned as the second or third symptom. Etiology and treatment are discussed. Theodore M. Shapira.

Fonseca, Aureliano. **Amaurotic family idiocy.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 343-351.

The author reviews briefly cases of this condition reported in Brazil. He records one more case in a child born in Brazil, whose ancestors four generations back were born in Germany.

Ramon Castroviejo.

Fonseca, Aureliano. **Ocular disturbances produced by rachianesthesia.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 353-354.

The author reports the case of a woman 35 years of age on whom a laparotomy was performed under rachianesthesia. A year after the operation very marked diminution of vision occurred, gradually leading to blindness of the right eye and counting fingers at six feet for the left eye. Ophthalmoscopy revealed marked atrophy of both optic nerves, and the author believes that this atrophy was produced by the rachianesthesia.

Ramon Castroviejo.

Giqueaux, R. E. **Hereditary atrophy of the papillomacular bundle.** Arch. de Oft. de Buenos Aires, 1938, v. 13, March, p. 111.

This article describes the occurrence of hereditary atrophy of the papillomacular bundle in a family. Points of resemblance and dissimilarity to Leber's optic atrophy are discussed in detail. Myopia of slight or moderate degree was found in most of those affected. The disease, occurring bilaterally, begins at an early age. It is characterized by pallor of the temporal segment of the nerve head and a cecocentral scotoma. Females are affected as well as males.

Edward P. Burch.

Kravitz, Daniel. **Studies of the visual fields in cases of verified tumor of the brain.** Arch. of Ophth., 1938, v. 20, Sept., pp. 437-470.

The findings as to 23 cases of verified tumor of the brain are presented in four groups: tumors of the frontal lobe, 6 cases; tumors in the region of the chiasm and the midbrain, 8 cases; tumors posterior to the chiasm, 6 cases; and tumors of the posterior fossa, 3 cases. The visual fields in the fourteen cases in groups 2 and 3, which might be expected to present localizing defects, showed signs of definite localizing value in 86 percent. From a diagnostic standpoint, a correct negative field may be as important to the neurosurgeon as a positive one. Of the nine cases of tumor in the frontal and posterior fossa eight, correctly, showed no localizing defect. The value of the visual fields is evident, since neurologic examinations gave correct results in only 70 percent of these cases. It is urged that every patient with persistent headache or other signs suggestive of possible tumor

should have an examination of the visual fields. J. Hewitt Judd.

Luzsa, Endre. **The influence of enucleation on the development of the optic canal.** Klin. M. f. Augenh., 1938, v. 101, Sept., p. 413.

In the living, the roentgenogram alone can demonstrate changes in the optic canal; and then only if compared with that obtained under equal circumstances for the other side. Luzsa studied this problem in 37 individuals, in each of which one eye had been enucleated—in four before the eighteenth year and in twenty after the twentieth year. In 15 further cases with enucleation between three and twenty years of age the canal on the side of enucleation was smaller than on the intact side, with one exception. The author found that the development of the optic canal terminated at about the eighteenth year. After enucleation before that time the development of the canal was retarded in consequence of atrophy of the optic nerve, and in the roentgenogram it appears narrow as compared with the healthy side.

C. Zimmermann.

Mueller, Friedrich. **Atrophy of the optic nerve of unknown etiology.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 329-330.

The author reports such a case.

Samuels, Bernard. **The histopathology of papilledema.** Amer. Jour. Ophth., 1938, v. 21, Nov., pp. 1242-1258.

Sloan, L. L., and Woods, A. C. **Perimetric studies in syphilitic optic neuropathies.** Arch. of Ophth., 1938, v. 20, Aug., pp. 201-253.

After a historical survey, the authors report the field defects in a group of 56 patients with primary syphilitic optic atrophy, which were of four separate types: (1) concentric contraction of the peripheral field associated with late loss of vision; (2) sector-shaped, or nerve-bundle, defects, with which loss of vision might be early or late, depending on the involvement of the papillomacular bundle; (3) central or cecocentral scotoma with normal peripheral fields, associated with early loss of visual acuity; and (4) central or cecocentral scotoma with defects in the peripheral fields, also associated with early loss of vision. Central or cecocentral scotoma was found in 53 percent of the cases of primary atrophy of the optic nerve. These studies indicate that perimetric changes may antedate either visual failure or pallor of the disc, and that the site of the lesion responsible for the field defects is in the optic nerve rather than in the chiasm or posterior to it and is probably a peripheral and interstitial neuritis with secondary degeneration. J. Hewitt Judd.

Vail, Derrick. **Optochiasmic arachnoiditis. Importance of a mixed type of atrophy of the optic nerve as a diagnostic sign.** Arch. of Ophth., 1938, v. 20, Sept., pp. 384-394.

Study of the cases reported in the literature, associated with the clinical findings in two personal cases reported in detail, suggests that the involvement of the optic nerve is a combination of papilledema and simple atrophy. One or the other may predominate and the resulting atrophy will take on a mixed character. This appearance, in which the outline of the disc is sharply defined and the lamina cribrosa is visible, but the caliber of the vessels is markedly reduced, is highly suggestive if not pathognomonic of arachnoiditis involving the chiasm. Recent evidence indicates that encephalitis, multiple

sclerosis, other demyelinating diseases, sinusitis or focal infections, and optochiasmic arachnoiditis may be closely allied factors in producing retrobulbar optic neuritis and may be different manifestations of a single pathogenic process such as thrombosis in the venous system of the nerve tissue. (Bibliography.) J. Hewitt Judd.

Vidal, J., and de Farias, N. **Treatment of optic-nerve atrophy by inoculation of malaria.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 325-327.

The authors obtained marked improvement of vision in a single case so treated. The treatment should include iodides and bismuth.

Ramon Castroviejo.

12

VISUAL TRACTS AND CENTERS

Custodis, Ernst. **Anatomic investigation and clinical observations on the syndrome of pterygopalatine-fossa lesions.** Zeit. f. Augenh., 1938, v. 95, Aug., p. 259.

Isolated synchronous and homonymous paralysis of the second ramus of the trigeminal nerve and of the abducens does not indicate a tumor of the pterygopalatine fossa. A tumor in this situation cannot involve these nerves by means of such pressure or infiltration without including the neighboring oculomotor nerves. The injury is caused by a malignant neoplasm which arises in the epipharynx and traverses the root of the pterygoid process. In the author's case, with further extension along the base of the cranium, destruction of the apex of the petrous portion of the temporal bone produced paralysis of the sixth nerve. Other fibers frequently involved are the secretory fibers of the lacrimal gland, the third branch of the trigeminus, and the sympathetic fibers.

F. Herbert Haessler.

Genet, L., and Rosnoblet. **Blindness without ophthalmoscopic signs in an infant; congenital form.** Bull. Soc. Franç. d'Opht., 1937, v. 50, pp. 240-246.

The authors report the case of a thirteenth-month-old infant whose parents had noted lack of vision when the infant was six months old. There had been no previous local or general diseases. The results of the physical and laboratory examinations were negative. There were no external or internal abnormalities of the eyes. There was only a slig[illegible]upillary response to light. Agenesis of the intracerebral optic pathways was considered as a possible cause. Clarence W. Rainey.

Oliva, Roberto. **Ocular symptoms in affections of the apex of the petrous portion of the temporal bone.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 357-360.

The author discusses briefly the relation between the fifth and sixth nerves, and the affections of the apex of the petrous bone. The symptoms that most directly interest the ophthalmologist are produced by paralysis of the abducens and neuralgia of the trigeminus, the first condition being known as the syndrome of Gradenigo. The author reports two cases, and urges ophthalmologists to be on the lookout for characteristic symptoms.

Ramon Castroviejo.

Paiva, Aroldo. **Suboccipital puncture from the ophthalmologic viewpoint in cranial injuries.** Arch. de Oft. de Buenos Aires, 1938, v. 13, Feb., p. 92.

Cisternal puncture for relief of in-

creased intracranial pressure complicating head injuries is advocated by the author. He believes it to be superior to lumbar puncture. A brief summary of four cases of head injury treated by suboccipital puncture is given. The author would extend this method of treatment to other types of intracranial hypertension which might give rise to optic-nerve changes.

Edward P. Burch.

Pesme, P., and Hirtz. **Observations of the syndrome of Laurence-Bardet-Biedl.** Bull. Soc. Franç. d'Opht., 1937, v. 50, pp. 297-310.

The authors report their observations of four cases, three males and one female, all under the age of sixteen years, who presented the picture of obesity of the hypophyseal type—genital hypoplasia, waxy pallor of the discs, ashy-gray changes at the macula, and peripheral retinal changes, described as retinal degeneration without pigment. The symptom of polydactyly was absent. The authors were able to obtain marked improvement in one patient by multiglandular therapy.

Clarence W. Rainey.

Wauters, Marcel. **A case of bilateral amaurosis following abortion.** Bull. Soc. Belge d'Opht., 1938, no. 76, p. 59.

The patient, 28 years of age, and three months pregnant, attempted abortion by intrauterine injection of a solution of soap. A few minutes after the injection she became unconscious and vomited. A few hours later, upon regaining consciousness in a hospital, she declared that she was totally blind. The same night she expelled two fetuses 5 cm. in length. The loss of blood was not severe. In a week her vision had returned to normal. The author attributed the amaurosis to intoxication caused by the absorption of soap by the uterine mucosa. In discussing the report Coppez stated his belief that the amaurosis had been simulated. (13 references.) J. B. Thomas.

13

EYEBALL AND ORBIT

Auffinger, Erwin. **Contributions to a solution of the problem of implantation of spheres after enucleation.** Zeit. f. Augenh., 1938, v. 95, Aug., p. 241.

Of 58 implants of bone, more than 49 were retained, and the result was considered satisfactory in all but eight of these. Nevertheless, for several reasons, the author discontinued the use of a bone sphere. The least of these was persistently irritated conjunctiva with copious tearing and mucopurulent exudate which might last for months. Sometimes the implant, although retained, became exposed through pressure atrophy and necrosis of the tissues. When a bone sphere had to be removed, it was found to be traversed by organized granulation tissue, and excision was extremely difficult. Local anesthesia was usually inadequate.

In six patients, the author implanted cartilage, but in four of them the sphere was extruded. In the end, spheres of amber were considered the most satisfactory. They are readily sterilized, are well tolerated by the tissues, resist absorption, and are easily made in any required size. A 15-mm. sphere weighs 1.5 gm. When amber spheres are extruded, they come away easily and cause no injury.

At the time of enucleation, the author threads all four rectus muscles on a single suture before cutting them. A special speculum with eight hooks helps keep the suture from becoming entangled. If this suture is tied as a

purse string, it forms a muscular stump which is useful even when the implant is lost. F. Herbert Haessler.

François, J. **Voluminous bony tumor of the external wall of the orbit, the great wing of the sphenoid, and the temporal fossa.** Bull. Soc. Franç. d'Opht., 1937, v. 50, pp. 281-296.

A 45-year-old woman presented herself because of progressive exophthalmos of the right eye and swelling in the right temporal fossa. There was X-ray evidence of a large bony tumor. No operation was done.

Clarence W. Rainey.

Friede, Reinhard. **A practical chalazion suture.** Graefe's Arch., 1938, v. 139, pt. 2, pp. 325.

Where bleeding persists after excision of the chalazion through the skin, the author recommends a double-armed suture at each side of the wound, passing through skin, orbicularis muscle, and tarsus, tightened and tied.

H. D. Lamb.

Groenouw. **A case of spontaneous orbital hemorrhage.** Klin. M. f. Augenh. 1938, v. 101, Sept., p. 420.

On March 8, 1938, a woman aged 61 years came on account of sudden amaurosis of the hitherto perfectly healthy left eye, with headache and vomiting. There was ecchymosis under the skin of the lids, and bloody discoloration of the lower half of the ocular conjunctiva; with exophthalmos upward and limited motility. The pupil did not react directly to light, but did consensually. The fundus was normal, but vision was limited to light perception. Two weeks later the lateral half of the optic disc was pale, and after six weeks more the whole disc was completely white and the visual field concentrically contracted. The condition was undoubtedly caused by a hemorrhage into the orbit due to a local affection of the orbital blood vessels.

C. Zimmermann.

Jaeger, Antoine de. **Notes on certain cases of infection of the orbit.** Bull. Soc. Belge d'Opht., 1938, no. 76, p. 63.

The writer classifies infections of the orbit according to Birch-Hirschfeld, as follows: (1) inflammatory edema of the orbit; (2) inflammation of the bony wall and periosteum of the orbit, especially in connection with sinusitis; (3) phlegmon of the orbit or orbital thrombophlebitis; (4) syphilis and tuberculosis of the orbit; and (5) tenonitis and pseudo-inflammatory tumors. Several case histories are reported illustrating these various types of infection. Two cases of retrobulbar phlegmon followed dacryocystorhinostomy done for acute dacryocystitis. In the discussion this procedure during the acute stage of a dacryocystitis was criticized, as was also simple extirpation of the sac in like circumstances. (4 X-ray plates). J. B. Thomas.

Krause, A. C., and Weekers, R. **Inositol in the ocular tissues.** Arch. of Ophth., 1938, v. 20, Aug., pp. 299-303.

Inositol was found in all tissues of the globe, with the possible exception of the vitreous. It was relatively constant in concentration in the different types of tissue but varied in different tissues. Concentration was high in the lens and in the optic nerve. It is thought that inositol may be a stable form of metabolite in the tissues which have no glycogen or only a small amount of it.

J. Hewitt Judd.

Kurz, Otto. **Blood cyst of the orbit.** Klin. M. f. Augenh., 1938, v. 101, Sept., p. 405.

A girl aged seventeen years had noticed three weeks previously a sudden swelling and blue discoloration of her left upper lid, with exophthalmos, pain, and diplopia. At the nasal side of the eyeball an elastic movable resistance could be felt. The optic disc was indistinct, about five diopters prominent, with peripapillary edema. and vision 6/12. A bluish cystic tumor was extirpated from the depth of the orbit. It was a hematoma with a wall of connective tissue and with a vascular convolution at its posterior end. It was interpreted as the product of a thrombus in the ectatic venous convolution, caused through stasis during strenuous work, to which adenoid vegetations may have contributed as a respiratory impediment. The eliciting factor may have been vasomotor insufficiency during the menstrual cycle.

C. Zimmermann.

Meyer, K., and Smyth, E. M. **On the nature of the ocular fluids. 2. The hexosamine content.** Amer. Jour. Ophth., 1938, v. 21, Oct., pp. 1083-1090.

Smelser, G. K. **Treatment of experimentally produced exophthalmos with thyroxin and other iodine compounds.** Amer. Jour. Ophth., 1938, v. 21, Nov., pp. 1208-1218.

14

EYELIDS AND LACRIMAL APPARATUS

Fazakas, Alexander. **Congenital symmetric ocular anomalies.** Klin. M. f. Augenh., 1938, v. 101, Aug., p. 257.

In investigating anomalies of the tear passages the author found numerous disturbances of development in the upper part of the lacrimal drainage tubes and around the whole palpebral opening. These he describes in detail.

C. Zimmermann.

Gernet, R. **Remarks on the Blaskovics ptosis operation.** Klin. M. f. Augenh., 1938, v. 101, Sept., p. 422.

The simplified method described removes the deformities without resorting to implantation of auricular cartilage, regarded by Blaskovics as the only possibility for correcting such conditions. C. Zimmermann.

Kreibig, Wilhelm. **Plastic reconstruction of the lids.** Zeit. f. Augenh., 1938., v. 95, Aug., p. 269.

To make a new upper lid, the author uses a large pedunculated flap whose base is in front of the ear and which extends forward over and to the inner end of the eyebrow. It is important that the end of the upper incision near the ear shall lie at the level of the upper edge of the eyelid.

F. Herbert Haessler.

Lijo Pavia, J. **Marked bilateral inflammatory ectropion.** Arch. de Oft. de Buenos Aires, 1938, v. 13, March, p. 149. (See Amer. Jour. Ophth., 1938, v. 21, Dec., p. 1423.)

Mirič, B. **A simple operation for entropion and trichiasis due to trachoma.** Klin. M. f. Augenh., 1938, v. 101, Sept., p. 381.

On account of the unsatisfactory results of the usual operations for entropion and trichiasis, the author devised a new method which in 161 cases gave good therapeutic and cosmetic results, and which is described in detail with illustrations. By stitching the separated upper part of the tarsus into the wound along the ciliary margin, the contracting effect of the levator is displaced to a lower point and its function increased, so that it supports aversion of the lid margin and cilia from the eyeball. C. Zimmermann.

Mohamed, I. A. **Trachoma of the lacrimal apparatus.** Bull. Ophth. Soc. Egypt, 1936, v. 29, p. 7.

Since the lacrimal apparatus normally contains lymphocytic adenoid elements, a simple chronic inflammation may easily be mistaken for a specific trachomatous inflammation. Histopathologic studies give inconclusive evidence as to whether or not trachoma primarily attacks the lacrimal apparatus. A careful review of the literature is given. Photomicrographs of cystic dilation of the ducts of the lacrimal gland ascribed to gradual incomplete or intermittent obstruction of the lacrimal outflow following cicatricial processes in the conjunctiva are shown, as well as several photographs of follicular dacryocystitis corresponding to all stages of trachoma.

Edna M. Reynolds.

Southgate, Paul. **A treatment for chronic dacryocystitis.** Amer. Jour. Ophth., 1938, v. 21, Oct., pp. 1158-1161.

15

TUMORS

Appelmans. **Treatment of epithelioma of the eyelids.** Bull. Soc. Belge d'Opht., 1938, no. 76, p. 75.

Basocellular or tubular epithelioma represents about 70 percent of these cases, the spino-cellular or lobulated form 15 percent, and the mixed form the remaining 15 percent. Epithelioma of the lid is preceded by a precancerous lesion in about 20 percent of all cases. The most important criterion for judging the gravity of a palpebral epithelioma is the age of the lesion, that is, its stage of evolution. The treatment described is that practiced by Maisin at the Cancer Institute of Louvain. Radium therapy is preferred and the results were happy in 134 out of a total of 150 cases thus treated. As to the permanence of these favorable results, 7 had remained cured for at least 10 years, 6 for 9 years, 3 for 8 years, and 5 for 7 years. It is admitted that cataract may follow in a certain percentage of cases in which treatment has been energetic or prolonged but in view of the gravity of the disease the radium treatment should not be condemned. In the problem of cancer one must bear in mind that the individual tumor is not all. The study of precancerous lesions and the observation of multiple cancers show that the general condition must have a part in the genesis of cancer. Radium cures the local lesion but the cancerous tendency remains, so these patients must be kept under observation. In discussing the paper, Hubin stated that he had abandoned radium in favor of diathermy coagulation, using the unipolar method (without indifferent electrode). Weekers noted that diathermy was more convenient, was less costly for the patient, and left a scar comparable with that of radium. He insisted on the importance of the oculist directing the treatment of these cases, assisted by the radiologist and dermatologist. (16 references.)

J. B. Thomas.

Binkley, G. W., and Motto, M. P. **Primary epibulbar prickle-cell epithelioma.** Amer. Jour. Ophth., 1938, v. 21, Oct., pp. 1156-1158.

Birge, H. L. **Cancer of the eyelids, conjunctiva, and cornea. 2. Squamous cell epithelioma.** Arch. of Ophth., 1938, v. 20, Aug., pp. 254-270.

This condition was found in 59 out of 230 cases of epithelioma. It originated from the conjunctival surfaces, including the cornea, in 37 percent. Primary

corneal involvement occurred in 5 percent. The malignancy of most of the lesions was either grade two or grade three. There was a close correlation between clinical behavior and grade of malignancy; and recurrences were frequent. The average mortality of all types was about 12 percent. The grade of malignancy should be determined first, and treatment proportional with this rating should be carried out in order to prevent recurrences. The article includes eight case reports and photographs of various types of the tumor. J. Hewitt Judd.

Capuano, Attilio Jose. **Contribution to the study of sarcoma of the choroid.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 281-287.

The author reviews some cases from the literature and reports one in a patient 42 years of age. Ophthalmoscopic examination of the eye seemed to show characteristics of endophthalmitis, but finally a diagnosis of sarcoma of the choroid was made. Microscopic examination revealed a spindle-cell sarcoma.
Ramon Castroviejo.

Dunnington, J. H. **Intraocular tension in cases of sarcoma of the choroid and ciliary body.** Arch. of Ophth., 1938, v. 20, Sept., pp. 359-363.

An analysis of 55 cases of intraocular sarcoma was made to determine the diagnostic value of intraocular tension in differential diagnosis between a serous detachment of the retina and one due to an intraocular tumor. In nine cases a definite secondary glaucoma had occurred. In 29 of the remaining 46 cases, the tension of the affected eye was found to be lower than that of the sound eye. In eleven the pressure was equal, while in six the affected eye showed a slightly higher tension. The decrease, which varied from 2 to 10 mm. of mercury, was found as frequently when the tumor originated in the choroid as when it started in the ciliary body. The conclusion is reached that an initial drop in intraocular tension is the rule rather than the exception in the early stages of sarcoma of the choroid and ciliary body.
J. Hewitt Judd.

François, J. **Aneurysmal dilatation of the ophthalmic artery.** (Melanosarcoma.) Bull. Soc. Franç. d'Opht., 1937, v. 50, pp. 264-266.

Previously the author had reported an aneurysmal dilatation of the ophthalmic artery, with superior pseudohemianopsia, reduction of the visual acuity to 0.5, papillary stasis, and mild hypertension of the cerebrospinal fluid. Operation had revealed involvement of the optic nerve near the optic foramen, with a gray violaceous mass which was the artery doubled in volume. Subsequently the vision diminished, the retina became detached, and the tension rose. In the enucleated eye a pea-sized melanosarcoma was found.
Clarence W. Rainey.

Kronenberg, Bernard. **Topography and frequency of complications of uveal sarcoma.** Arch. of Ophth., 1938, v. 20, Aug., pp. 290-298.

Most tumors are localized posteriorly and temporally. Eighty-eight percent of the 126 cases of uveal sarcomas were located in the choroid. There was an equal distribution between the two sexes and the average age was 52.6 years. The predominant type was the spindle-cell sarcoma. Almost all circumpapillary sarcomas caused detachment of the retina, and this complication occurred in more than 75 percent of all of these cases. The topographic

disturbance does not influence the frequency of glaucoma, which was found to be present in half of the eyes of this series. The site of the sarcoma has no bearing on the frequency of extraocular extension, which occurred in 36.1 percent. J. Hewitt Judd.

Lijo Pavia, J. **Primary sarcoma of the choroid; early diagnosis; enucleation of eye having normal vision.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 276-279.

In a patient 27 years of age a small growth was found near the left macula. Visual fields taken at short intervals after the first examination showed the blind area to be growing larger. Microscopic examination of the enucleated eye proved the clinical diagnosis to be correct, the tumor being a melanosarcoma of the choroid.

Ramon Castroviejo.

Mathis, G. **A case of leucosarcoma of the choroid.** Rassegna Ital. d'Ottal., 1938, v. 7., May-June, pp. 408-415.

The patient was a man of 71 years whose left eye had been blind for four years and had previously been treated for glaucoma. A small mass was seen in the anterior chamber in the lower nasal quadrant, at the root of the iris. Upon removal of the eye a mass was found on the outside of the globe, at the corresponding point, measuring 13 by 15 by 5 mm. When the globe was sectioned a tumor mass was found at the base of the iris, measuring 10 by 12 mm. Thus the extrabulbar portion of the tumor was greater than the intraocular. The cells were polymorphous and no pigment was found in them. At the point of perforation the scleral lamellae were widely separated. (3 figures.) Eugene M. Blake.

Mueller, F. **Extirpation of a carcinoma of the lower eyelid and plastic repair of the defect.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 249-250.

The defect was covered with sliding skin flaps.

Stallard, H. B. **A new technique for the application of radon seeds to the sclera in the treatment of glioma retinae.** Brit. Jour. Ophth., 1938, v. 22, Oct., pp. 604-608.

The technique described varies from that of Foster Moore as used at St. Bartholomew's Hospital. The cases of two children in whom the glioma was adjacent to the optic disc on the temporal side are described. In each case no increased irritation, no infection, and no sepsis followed the procedure., and there was no evidence of damage to the optic-nerve head or to the ocular media. The method is said to be technically simple and to be effective and free from complications. The radon seeds are embedded in a strip of dental stent shaped to the sclera at the site, and secured in place by sutures traversing the superficial layers of the sclera in front of and behind the equator. (Figures, references.)

D. F. Harbridge.

16

INJURIES

Agnello, Francesco. **Nasal syndrome and neuralgia of trigeminus through cornea injury.** Riv. Oto-Neuro-Oft., 1938, v. 15, Jan.-Feb., pp. 79-86.

A woman of forty years after a wound of the left cornea by her fingernail, became affected by intense pain around the corresponding orbit. The pain did not yield to the action of sedatives or hypnotics. The cornea showed

a linear wound whose character was not proportioned to the intensity of the other symptoms. A woman of 36 years was wounded on the left cornea by the fingernail of a baby. The same recurrent symptoms followed. In each the symptoms disappeared after treatment given to improve the general condition of the patient. M. Lombardo.

Alexandrov, V. V. **The rationale of the set-up for the campaign against ocular traumatism.** Viestnik Opht., 1938, v. 12, pt. 2, p. 271.

A description of the official set-up of this department of ocular prophylaxis.

Ray K. Daily.

Aliquò-Mazzei, Alessandro. **Treatment of traumatic rupture of the sclera.** Boll. d'Ocul., 1938, v. 17, Feb., pp. 80-91.

Of four cases of indirect or contused rupture of the sclera, three were caused by the horn of an ox in farm workers. The fourth case was in a mechanic aged 19 years whose eye was struck by a fragment of a firearm. All four patients showed subconjunctival rupture near the limbus in the upper nasal segment of the globe, with subconjunctival dislocation of the lens. Conservative treatment is to be used, and enucleation or evisceration should not be resorted to sooner than a month after the injury. Surgical and prophylactic treatment are discussed. (Bibliography.)

Melchiore Lombardo.

Avalos, Enrique. **Electric ophthalmia as an industrial accident.** Rev. Cubana Oto-Neuro-Oft., 1938, v. 7, March-April, p. 41.

The effects upon the eye of electrical energy of varying intensities are described, and prophylactic measures suitable for workers exposed to such harmful effects are proposed.

Edward P. Burch.

Baltain, M. M. **X-ray localization of intraocular foreign bodies.** Viestnik Opht., 1938, v. 16, pt. 1, p. 81.

A review of the various methods in use. The author prefers Comberg's method, in the use of which he replaces the contact glass by a prothesis of his own design. In doubtfully localized foreign bodies, he uses injection of radiopaque substances into Tenon's capsule. (Illustrations.)

Ray K. Daily.

Georgariou, P. M. **Cholesterinosis bulbi.** Graefe's Arch., 1938, v. 139, pt. 1, pp. 32-49.

A girl 14 years old, who had been struck in the left eye with scissors ten years previously, had been totally blind for two years. The eyeball had recently shown much ciliary and conjunctival injection and a filling of the anterior chamber with a dense, white compact mass of rhombic cholesterine crystals. By paracentesis, these crystals were drawn off and their character confirmed. Thereafter the intraocular tension remained very low. Cholesterine crystals appeared again in the anterior chamber. This condition, to be distinguished from lipoidosis bulbi, is here described for the first time.

H. D. Lamb.

Gonçalves, Paiva. **Penetrating orbitocranial injury produced by fencing saber without lesion of skin or eyeball, and retention of metallic fragment in posterior cerebral fossa.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 237-248.

This trauma was sustained by an officer of the army, 29 years of age, during

a fencing lesson. The saber of his opponent entered the left orbital cavity through the conjunctiva, nasally to the eyeball, without injury to the globe itself. The injury was followed by slow pulse, vomiting, slight rise of temperature, severe headache, and semicoma. Locally there was marked chemosis of the eyelids, as well as subconjunctival hemorrhage more pronounced near the inner canthus. The eye was slightly deviated downward and outward. The fundus was normal. X ray showed to front view a foreign body which seemed to be located near the apex of the orbital cavity. The patient was operated upon under local anesthesia, an incision being made in the skin of the upper lid near the orbital margin, and over the nose. No foreign body was found, and the walls of the orbit appeared to be normal. A second X ray, showing a profile of the skull, revealed a foreign body located within the cranial cavity in the left occipital region. The cranial cavity was exposed by trephining a small flap of bone in the left occipital region, and a metallic foreign body 5 by 1 mm. was found near the opening and was easily removed. In a few months both eyes had normal motility as well as normal vision. Reconstruction of the path of the foreign body showed that the saber must have entered the left orbit through the conjunctiva near the inner canthus, penetrated the tendinous ring of Zinn, transversed the sphenoidal fissure, and passed through the whole cranial cavity to strike the occipital bone, where the point of the saber remained after the saber was withdrawn.

Ramon Castroviejo.

Grancini, L. E. **Contribution to the study of ocular lesions from electric discharge, especially in relation to lens changes.** Boll. d'Ocul., 1938, v. 17, Feb., pp. 92-111.

The face of a man of 45 years was swept by the flame developed from contact of an iron tool with a high tension wire. No shock was received. A few hours later his left cornea was slightly turbid, the iris hyperemic, the pupil slightly dilated and not reacting to light. The lens had a posterior capsular cataract and tension was 85 mm. (Schiötz.) Lens injury occurred in a man of 39 years, who on raising a hammer to within about 30 cm. of a high tension wire received a shock from which he remained unconscious for about six hours. The writer reviews experimental researches on electric cataract, and comes to the following conclusions: Electric cataract is the direct and immediate effect of the passage of the electric current through the lens. A co-existing uveal lesion favors formation of cataract. The cataract may reach maturity or the lens may remain partially transparent. (Bibliography.)

Melchiore Lombardo.

Hertel, E. **The closure of the wound by operation in perforating injuries of the cornea.** Graefe's Arch., 1938, v. 139, pt. 1, pp. 1-16.

The author agrees with Kuhnt that where there is a central, straight, perforating corneal wound with smooth sides and of an obliquity up to 3 mm., in the absence of conjunctivitis or tear-sac disease, no operative procedure is indicated. In the overwhelming majority of the author's cases, however, there was present a large wound with gaping and partly displaced wound edges and frequent prolapse of the iris. Operation was then necessary to close the wound and consisted of corneal suture or conjunctival flap, or both. In corneal suture, the thread does not pene-

trate the entire thickness of the cornea but only to the junction of its middle and posterior thirds. Nine illustrations taken from sections through healing corneal wounds, clearly show the approximation of the wound edges obtained by corneal suture or conjunctival flap. H. D. Lamb.

Kamel, Sabri. **A case of traumatic total depigmentation of the iris.** Bull. Ophth. Soc. Egypt, 1936, v. 29, p. 156.

A case is reported in which large patches of iris pigment were scattered over the lens capsule, together with hundreds of minute pigment granules. There was a superficial opacity of the lens. The iris meshes were entirely normal and the pupil showed no synechiae. The iris was tremulous and vision was reduced to hand movements. These changes occurred within 24 hours after injury. Edna M. Reynolds.

Lazarev, E. G. **A new system of protective lenses for workers.** Viestnik Opht., 1938., v. 12, pt. 2, p. 269.

The author describes a new design which consists principally of stenopeic slits, placed horizontally in one eye and vertically in the other. (Illustrations.) Ray K. Daily.

Medvedev, H. I. **So-called consensual ocular reactions.** Viestnik Opht., 1938, v. 12, pt. 3, p. 338.

This is a report of tonometric curves of both eyes in 46 cases of traumatism to one eye. The conclusions are that the primary reaction to traumatism may be either hypotension or hypertension, depending on the complications. There is a reflex effect on the uninjured eye, the extent and duration of which depends on the reaction of the diseased eye. The most marked feature of the reflex changes is the increased amplitude of the daily tension curve. There may also be an effect on the adaptation, corneal sensitivity, angioscotomata, and accommodation. Ray K. Daily.

Rapisardo, Dante. **A rare case of detachment of the pars iridica retinae.** Boll. d'Ocul., 1938, v. 17, Jan., pp. 40-44.

A man of 23 years who had sustained a perforating wound of the cornea at the age of nine years showed a dense adherent leucoma at the 5-o'clock position. Lens capsule was incarcerated, and there was posterior synechia with adhesion of the upper part of the iris to the lens capsule. The pupillary area was in great part occupied by a membrane of the same color as the pupil. This formation evidently consisted of posterior layers of the iris which had become adherent to the lens capsule and had been brought down and held in that position by the part of the capsule included in the corneal scar. Melchiore Lombardo.

Sherman, G. C. **Ocular injury with an indelible pencil.** Viestnik Opht., 1938., v. 12, pt. 2, p. 277.

A six-year-old girl came to the clinic six days after perforating the right eye with an indelible pencil, a particle of which remained in the eyeball. The aqueous, the lens capsule, and the iris had a violet tint, the fundus was invisible, and vision was counting of fingers close to the face. The anterior chamber was opened and was irrigated with 1 to 1000 tannic-acid solution, and two drops of 1 to 1000 collargol were introduced. A fibrinous iridocyclitis followed, and the result was a quiet eye with an opaque cornea, and vision equal to light perception. Ray K. Daily.

Vila Ortiz, J. M. **Case of traumatic cataract in rosette.** Trabalhos do Prim-

eiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 249-251.

The author has already reported five such cases, in all of which he found a history of trauma. The present patient had sustained a severe blow on the head 25 years previously.

Ramon Castroviejo.

Wiegmann, E. **A case of spontaneous reattachment of a traumatic iridodialysis.** Klin. M. f. Augenh., 1938, v. 101, Sept., p. 423.

In a laborer a small traumatic iridodialysis became spontaneously reattached, showing that in small dialyses an expectant treatment may be indicated. C. Zimmermann.

Wölfflin, E. **On the time relationship of the occurrence of lens opacities after radiation with various wave lengths.** Klin. M. f. Augenh., 1938, v. 101, Sept., p. 321.

The varying time of occurrence of lens opacities after radiation with various wave lengths is not a uniform process which can be explained on a purely physical basis. The action of infrared and ultraviolet is a direct damage by absorption. Injuries by roentgen and radium rays are probably caused indirectly through the nourishing organ, namely the ciliary body. Otherwise the long latent period would not be physiologically conceivable, nor would it be logical that the most posterior parts of the lens should first become opaque. C. Zimmermann.

Woodruff, H. W. **Spontaneous extrusion of an extraocular foreign body (shot) with recovery of vision.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 1028-1029.

17

SYSTEMIC DISEASES AND PARASITES

Andrade, Cesario de. **Complexity of the cervical oculo-sympathetic syndrome.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 361-366.

Mention is made of the different ocular symptoms that take place in affections of the cervical sympathetic. Three cases are reported: one in which after an alcohol injection of the phrenic nerve the patient developed a typical Horner syndrome; one of trauma to the cervical vertebrae, with a syndrome of the posterior cervical sympathetic, including marked myosis and enophthalmos; a third in a patient with a tumor of the throat causing irritation of the cervical sympathetic with exophthalmos and marked mydriasis.

Ramon Castroviejo.

Busacca, Archimede. **Worms and other intestinal parasites as cause of ocular affections, particularly uveitis.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 289-290.

Intestinal parasites may produce a number of ocular manifestations, namely blepharitis, keratitis, conjunctivitis, uveitis, retrobulbar neuritis, and papillitis. Elimination of the parasites invariably cures the ocular manifestations. Ramon Castroviejo.

Chojnacki, P. **Experimental and clinical studies on focal infection of dental origin.** Graefe's Arch., 1938, v. 139, pt. 2, pp. 288-300.

In ten cases of iritis and four of axial optic neuritis (two bilateral), one or more teeth with dead pulp were extracted. Cultures from the apices of the affected teeth showed in nine cases streptococcus viridans and in four cases

forms of streptococci other than viridans and hemolyticus. From 24-hour-old primary bouillon cultures in each case, intravenous injections were made into either two or four rabbits. Some degree of ocular infection followed in 24 rabbits and streptococci were isolated from the blood of 21 rabbits out of 37 inoculated. From an experience of over 25 cases of ocular disease due to dental infection, the author concluded that negative roentgen-ray findings in the teeth were not decisive and that a dead tooth root without a granuloma was much more dangerous than one with a granuloma. H. D. Lamb.

Dejean, C. **Fundus changes in the rabbit in rabies.** Bull. Soc. Franç. d'Opht., 1937, v. 50, pp. 247-154.

Twenty rabbits were inoculated with rabies through a trephine hole in the skull. Phenomena observed were: loss of corneal reflexes and pupil reactions to light; paralysis of the lid muscles, and of the external muscles of the globe; loss of transparency of the cornea and vitreous; intense congestion of the optic nerve and of the retina. The retina offers a true picture of the intracranial changes in rabies.

Clarence W. Rainey.

Eissa, A. S., and Kamel, Sabri. **Three cases of tuberculosis of the eye.** Bull. Ophth., Soc. Egypt, 1936, v. 29, p. 188.

The first case reported is one of primary tuberculous conjunctivitis and dacryoadenitis in a girl of seventeen years, which improved under general treatment. The second case is one of conglomerate tuberculous choroiditis which perforated the sclera near the limbus. The patient, aged five years, was found to be entirely free from tuberculosis elsewhere.The third case is one of cold abscess of the globe in a child of five years, who had pulmonary tuberculosis and died shortly after excision of the globe.

Edna M. Reynolds.

Gabriélidés, C. A. **Filaria in the anterior chamber of the eye.** Ann.d'Ocul., 1938, v. 175, Aug., pp. 581-589.

Report of one case of filaria in the anterior chamber. Instillation of pilocarpine in an attempt to lock the parasite in front of the iris seemed to irritate it, and the organism was lost behind the pupil. Enucleation was then performed and the parasite identified as a filaria.

John M. McLean.

Hermans. **The pathology of ocular affections of dental origin.** Bull. Soc. Franç. d'Opht., 1937, v. 50, p. 155.

The reports of Fromaget, 1924, and Worms and Bercher, 1925, are reviewed. The author gives a detailed theoretical discussion of the manner in which various dental abnormalities and infections may cause ocular disease.

Clarence W. Rainey.

Klivanskaja, A. A. **The eye as an indicator of congenital and acquired changes in children.** Viestnik Opht., 1938, v. 12, pt. 1, p. 116.

The author describes in detail the ocular symptoms of congenital syphilis, and hopes that very complete routine physical examinations of children will serve to detect, and to institute early treatment of, this disease.

Ray K. Daily.

Kreibig, W. **Blood diseases and the eye.** Wien. med. Woch., 1938, July 11, p. 772.

Kreibig describes the eye findings in various diseases of the blood-forming organs, including the anemias, throm-

bosis, acute hemorrhage, leukemias, polycythemia, and agranulocytosis.

Theodore M. Shapira.

Meyer, F. W. **Contribution to the syphilitic diseases of the eye.** Klin. M. f. Augenh., 1938, v. 101, Sept., p. 390.

A woman of 39 years was sent to a sanatorium with the diagnosis of right-sided scleritis and tuberculous keratitis. After a transfusion, she died suddenly from a thrombus in the carotid, which was mobilized by the transfusion and so induced embolism of the middle meningeal artery and cerebral hemorrhage. The autopsy revealed syphilitic mesaortitis and almost complete obstruction of the carotids, in connection with the roentgenological pulmonary findings, attributed to a luetic infection of about 10 or 15 years previously. But, the scleritis spoke for tuberculosis. The histologic changes in the eye are described in detail. The case shows the importance of seroreactions to lues, even in the presence of apparent ocular tuberculosis. C. Zimmermann.

Rosenblum, M. E. **Our latest cases of cysticercus.** Viestnik Opht., 1938, v. 12, pt. 3, p. 349.

Brief case reports of thirteen cases treated during the last five years. Of these eight were subretinal, two preretinal, and three in the vitreous. In three of the subretinal cases the cyst was in the region of the macula. General examinations were made in nine patients; tenia solium was found in four, and eosinophilia in six cases. Localization was by the method of Gonin, verified by electrocoagulation under ophthalmoscopic control. All cases were operated upon; in eleven the cysticercus was extracted, and in two the attempt failed. Ray K. Daily.

Safar, K. **Diabetes and the eye.** Wien. med. Woch., 1938, June 18, p. 685.

Safar discusses the various disturbances and changes that take place in the eye in diabetes. He states that symptoms are due only partly to disturbance in metabolism, blood-vessel changes also playing a part.

Theodore M. Shapira.

Schartz, S. E. **Carbon-monoxide poisoning.** Viestnik Opht., 1938, v. 16, pt. 3, p. 370.

The symptoms of carbon-monoxide poisoning are very bizarre generally as well as relative to ocular manifestations. The author's case was unusually severe, with edema and ecchymosis of the lids, chemosis, exophthalmos, limitation of movement, superficial keratitis, and rise of intraocular tension. The final result was loss of vision in the right eye and recovery in the left.

Ray K. Daily.

Simoes, Elyseo. **Heredity in ophthalmology.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 385-401.

After a long discussion on hereditary factors which play a role in ocular manifestations, the author reports six observations. The first is of a family of eight members, all having cataract. The second is of a small family in which there were four cases of infantile glaucoma. Consanguinity was found in the second instance. In the third family, of five persons, all had dyschromatopsia. In the fourth family, of 28 members in three generations, eight cases of albinism were observed. In the fifth family, 32 persons of three generations, there were twelve cases of albinism, and a history of consanguinity. The sixth family was of five persons in

which both parents and children were affected with myopia. The author concludes that there is a definite influence of heredity in ocular affections, consanguinity playing an important part in many cases. (63 references.)

Ramon Castroviejo.

Walker, J. R., and Walker, B. F. **A specific treatment for herpes zoster.** Arch. of Ophth., 1938, v. 20, Aug., pp. 304-306.

The authors report that diphtheria antitoxin has proved to be a specific remedy for herpes zoster in the acute as well as in the chronic stage, usually giving relief from pain within 24 hours and preventing recurrences.

J. Hewitt Judd.

Weckert, Fritz. **Biology in ophthalmology.** Graefe's Arch., 1938, v. 139, pt. 2, pp. 280-287.

A tuberculous inflammation of the retina or choroid resulting from bacillary emboli derived from the lymph glands at the hilus of the lung is an attempt with renewed energy to restore the normal state. Tuberculous and luetic infections of retina, uvea, and cornea correspond to similar infections of the cutis and the subcutaneous tissue of the skin. H. D. Lamb.

Zobel. **The treatment of headache and neuralgia by the oculist.** Zeit. f. Augenh., 1938, v. 95, June, p. 137.

The oculist need not send a patient away after prescribing glasses and determining that there is no cause for headache manifest in the fundus. Most headaches have their origin in a neuralgia of the trigeminal nerve. If pressure over the superior orbital margin causes pain or if the swollen nerve is palpable, the diagnosis of supraorbital neuralgia is certain. The author's treatment consists of application of a galvanic current of two milliamperes for five minutes to each branch of the nerve. For supraorbital neuralgia the cathode is placed on the inner end of the superior orbital margin, the anode on the neck. A metallic taste confirms the passage of current through the nerve. Five daily sessions usually cure the patient completely.

F. Herbert Haessler.

Zolotnitskii, I. H. **Malarial ocular complications in the Kiev region during the summer of 1935.** Viestnik Opht., 1938, v. 12, pt. 3, p. 377.

There were 68 cases classified as follows: keratitis herpetica 35, parenchymatous keratitis 2, herpes palpebralis 4, retinal hemorrhage 3, iritis 2, optic atrophy 1, dendritic keratitis 11, episcleritis 1, cyclitis 3, optic neuritis 1, convergent strabismus 1, vitreous opacities 4. The author urges that malaria be thought of in cases of obscure etiology, and that the melano-flocculation reaction be used in cases negative for the plasmodium. Ray K. Daily.

18

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Allen, T. D. **The value of routine in examinations of the eye.** Amer. Jour. Ophth., 1938, v. 21, Oct., pp. 1147-1152.

Bartels, Martin. **Schools for individuals with defective vision.** Klin. M. f. Augenh., 1938, v. 101, Aug., p. 161.

Examination of inmates of blind asylums revealed quite a number of children who were not blind but only had defective vision. The author was instrumental in the foundation of schools for children with defective vision. He shows the possibility of educating such children to useful callings, discusses the

equipment and instruction in these schools, and advocates an international system. C. Zimmermann.

Davis, W. T. **High lights of the history of ophthalmology.** Southern Med. Jour., 1938, June, p. 685.

This is an account of the development of ophthalmology from the time of the Code of Hammurabi to the present. John C. Long.

Esser, A. A. M. **Old Indian ophthalmology.** Klin. M. f. Augenh., 1938, v. 101, Aug., p. 263.

A historical essay.

Grolman, Gunther von. **The international prophylaxis of trachoma in our country (Argentina).** Arch de Oft. de Buenos Aires, 1938, v. 13, March, p. 129; also Rev. Oto-Neuro-Oft., 1938, v. 13, April, p. xxvii.

This article deals with regulations and problems regarding the inspection of immigrants to the Argentine for the purpose of excluding those suffering from trachoma. The chief difficulty is in arriving at a satisfactory and fair decision as to which individuals afflicted with trachoma grade 4 are capable of transmitting the disease.

Edward P. Burch.

Hitz, J. B. **An evaluation of visual-testing methods in schools.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 1024-1027.

Laskin, B. H. **Sanitary education in the campaign against ocular traumatism.** Viestnik Opht., 1938, v. 12, pt. 1, p. 122.

An outline of lectures on ocular sanitation, for groups of workers in industrial establishments. Ray K. Daily.

Leydhecker, F. K. **Concerning the eye, from Rückert's "Wisdom of the Brahmans."** Klin. M. f. Augenh., 1938, v. 101, Aug., p. 271.

Quotations from a didactic poem on personal and general questions of life.

C. Zimmermann.

MacCallan, A. F. **The world-wide distribution of trachoma, excluding the Dominions, Colonies, and mandated territories of Great Britain.** Brit. Jour. Ophth., 1938, v. 22, Sept., pp. 513-541.

It is the purpose of the author in this lengthy presentation to give an approximate estimate of trachomatization throughout the world, exceptions being noted which have been previously outlined (see Amer. Jour. Ophth., 1935, v. 18, March, p. 301). The use of the slitlamp not being possible in distant sections of the world, there must of necessity be an underestimate of trachoma actually existing in such regions. The article does not lend itself to abstract. D. F. Harbridge.

Mendoza, Rafael. **The oculist and industrial accidents.** Rev. Cubana de Oto-Neuro-Oft., 1938, v. 7, March-April, p. 49.

The author cites some of the difficulties encountered in industrial ophthalmology. Edward P. Burch.

Meyerhof, Max. **The history of trachoma treatment in antiquity and during the Arabic middle ages.** Bull. Ophth. Soc. Egypt, 1936, v. 29, p. 26.

The Greek medical works which have come down to us show a knowledge of four stages of trachoma, of entropion and trichiasis but not of pannus. The Greeks knew different scraping manipulations not much inferior to those in use at present. As a treatment for trichiasis they excised the skin but not the tarsus, and made an intermarginal incision with transposition of the lashes.

The Arabs knew the connection of trichiasis and pannus with trachoma, and appreciated its contagious character. Their mechanical and operative treatment of trachoma was not very different from our own. The best description of trachoma and its treatment before the 19th century was written by Ali ibn Isa, an oculist of Bagdad in the tenth century. Edna M. Reynolds.

Schwichtenberg, A. H. **The evaluation of orthoptic training for aviators.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 980-990.

Sena, J. A. **Some notes on the International Congress of Ophthalmology.** Arch. de Oft. de Buenos Aires, 1938, v. 13, March, p. 135.

The author discourses briefly upon the ocular disorders, chiefly trachoma, encountered in Egypt, giving a brief account of the ophthalmic hospital facilities. He also gives a résumé of the meeting of the International Organization Against Trachoma.

Edward P. Burch.

Vila Ortiz. **Instruction and professional orientation of the visually defective. Creation of special schools.** Arch. de Oft. de Buenos Aires, 1935, v. 13, Feb., p. 71.

The author makes a plea for the establishment of schools for the visually handicapped in the Argentine. He believes that children whose vision is 2/10 or less in the better eye with or without correction would be greatly benefited by the establishment of such schools. The selection of students and instructors, and the method and conditions of instruction are outlined in some detail. Edward P. Burch.

19

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Fortin, E. P. **The foveal mosaic.** Arch. de Oft. de Buenos Aires, 1938, v. 19, Feb., p. 51.

The author feels that our knowledge of the structure of the fovea based on histologic studies should be modified in the light of entoptic observations. He believes that in the foveal region there are specialized cones concerned chiefly with ocular fixation. These are 75 microns long and 2 in diameter and bear no resemblance to ordinary cones. (Illustrations.) Edward P. Burch.

NEWS ITEMS

Edited by Dr. H. Rommel Hildreth
640 S. Kingshighway, Saint Louis

News items should reach the Editor by the twelfth of the month

Deaths

Dr. Alfred F. VanHorn, Plainfield, New Jersey, died September 30, 1938, aged 77 years.

Dr. Harry S. Hughes, Saint Louis, Missouri, died September 15, 1938, aged 56 years.

Dr. Anders G. Hovde, Los Angeles, California, died October 14, 1938, aged 62 years.

Dr. Frank Clifford Ard, Westfield, New York, died August 23, 1938, aged 74 years.

Dr. Allen Rupert Cunningham, Halifax, Nova Scotia, Canada, died July 3, 1938, aged 50 years.

Dr. Harry Sebastian Reger, Jamestown, New York, died July 14, 1938, aged 60 years.

Miscellaneous

The American Board of Ophthalmology announces an important change in its method of examination of candidates for the Board's certificate. Written examinations will be held in various cities two months prior to the date of the oral examination. The first written examination will be on March 15, 1939. For further particulars see the editorial on page 85 of this issue of the American Journal of Ophthalmology.

Examinations will be divided into two parts. Candidates whose applications are accepted will be required to pass a written examination which will be held simultaneously in various cities throughout the country approximately 60 days prior to the date of the oral examination.

The written examination will include all of the subjects previously covered by the practical and oral examinations.

Oral examinations will be held at the time and place of the meeting of the American Medical Association and of the American Academy of Ophthalmology and Otolaryngology, and occasionally in connection with other important medical meetings. The oral examination will be on the following subjects: External diseases, Ophthalmoscopy, Pathology, Refraction, Ocular motility, Practical surgery.

Only those candidates who pass the written examination and who have presented satisfactory case reports will be permitted to appear for the oral examination.

Examinations scheduled for 1939. Written: March 15th and August 5th. Oral: Saint Louis, May 15th; Chicago, October 6th.

Applications for permission to take the written examination March 15th must be filed with the Secretary not later than February 15th.

Application forms and detailed information should be secured at once from Dr. John Green, Secretary, 6830 Waterman Avenue, Saint Louis, Missouri.

The University of Buffalo awards annually a gold medal for a work on an ophthalmologic subject. Recent awards went to Dr. J. Bellows of Chicago, Dr. Joseph Globus of New York, and Dr. J. Evans of Brooklyn. For details write to Dr. H. W. Cowper, 543 Franklin Street, Buffalo, New York.

A quarterly journal, The Journal of Social Ophthalmology, is now being published by the International Association for the Prevention of Blindness, with articles appearing in English and French. The address of the American office is 50 West 50th Street, New York City, with Lewis H. Carris the American correspondent.

The following new members were elected to fill vacancies on the Board of Directors of the National Society for the Prevention of Blindness, at the annual meeting of the Society, December 1, 1938: Dr. Thomas Johnson, ophthalmologist, New York City; Dr. John L. Rice, Health Commissioner of New York City; Professor Ira V. Hiscock, of New Haven, Connecticut, President of the National Health Council and Professor of Public Health at Yale University; Reverend John Gass, D.D., Rector of the Church of the Incarnation, New York City; Mrs. Hazel C. McIntire, of Columbus, Ohio, Director of Special Classes for the Visually Handicapped, Ohio State Department of Education. The following members of the Board of Directors were reëlected for a three-year period at the annual meeting: Dr. A. J. Chesley, St. Paul, Minnesota; Mr. George C. Clark, New York City; Dr. J. Clifton Edgar, Greenwich, Connecticut; Dr. Edward Jackson, Denver, Colorado; Dr. Albert B. Meredith, Cranford, New Jersey; Mr. Preston S. Millar, New York City; The Rev. Alphonse M. Schwitalla, Saint Louis, Missouri; Dr. William Zentmayer, Philadelphia, Pennsylvania.

Portland, Oregon, is to have a Medical Library Building. In Science (issue of November 25, 1938) we notice that Dr. John E. Weeks has given $100,000.00 to the University of Oregon Medical School. To this has been added an equal amount by the Rockefeller Foundation

and a sum secured from the Public Works Administration. These make a total of $363,350.00, to construct a building for the Medical Library, with an auditorium to seat 600 for student and professional gatherings.

Harvard Medical School offers the following courses in Ophthalmology for the first half of 1939:

February 6-18. Ocular Muscles. This course includes neuro-anatomy and physiology of ocular muscles as an introduction to didactic and clinical work. It deals extensively with vertical deviations. Orthoptics not included. This course is given by Drs. Bielschowsky and Casten.

March 1-31. A course on The use of the slitlamp will be given by Dr. Betham; a course on External diseases of the eye by Dr. Gundersen; and a course on Ocular complications in general disease by Dr. King. These courses may be taken simultaneously.

April 10-May 6. Recent Advances in Ophthalmology. This course correlates pathology of the eye and clinical practice. It draws on all resources of the Eye Department of the Massachusetts Eye and Ear Infirmary; clinical, laboratory, and research. Although this course has pathology as a basis, it gives a cross-section of all work at the Infirmary. The pathology is given by Dr. Terry.

July. Visual Optics and physiology. This course is given by Drs. Ludvigh, Cogan, and Easton.

Beginners and general practitioners may take the course in Ocular complications in general diseases. All other courses listed are not open to beginners. Further information may be obtained from the Assistant Dean of the Medical School.

Societies

The Eye Section of the Philadelphia County Medical Society met on December 1, 1938. Dr. Aaron Brav presented Trachomatous ulceration of the cornea, treated with prontosil. Dr. Temple Fay presented a moving-picture demonstration, in colors, Visual fields and other findings, including details of operation, and postoperative recovery of brain tumor. Dr. Walter I. Lillie discussed Dr. Fay's presentation.

The Canadian Ophthalmological Society, as newly instituted, held its first meeting in Montreal on August 24-25, 1938. The first president of the Society is Dr. Gordon Byers.

Personals

Dr. Daniel B. Kirby, surgeon, and Dr. A. Egerhoffer, senior resident at the Institute of Ophthalmology of the Presbyterian Hospital in New York, journeyed to Santa Fe, New Mexico, to operate on a number of patients with eye diseases at the Procter Memorial Eye Clinic, coöperating with Dr. W. C. Barton of Santa Fe. The operations included a great number of cataract, glaucoma, strabismus, and pterygia cases. The clinic was conducted through the month of August, 1938.

Dr. William R. Fringer announces that his son, Dr. Robert C. Fringer, is now associated with him in the practice of ophthalmology, William Brown Building, Rockford, Illinois.

Dr. C. W. Rutherford, Indianapolis, was the guest speaker at the meeting in Toledo, Ohio, Eye, Ear, Nose, and Throat Club the evening of November 17th.

Dr. William Thornwall Davis, Professor of Ophthalmology, George Washington University School of Medicine, Washington, D.C., has been invited to participate in the Continuation Study Course at the University of Minnesota on January 16, 1939. Dr. Davis will lecture on Vertical muscle imbalance in its relation to convergent squint, and the Choice of procedure in muscle surgery.